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ENCYCLOPÆDIA MEDICA

UNDER THE GENERAL EDITORSHIP OF
CHALMERS WATSON, M.B., M.R.C.P.E.

VOLUME II

BRACHIAL PLEXUS TO DIGESTION

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THE AUTHORS OF THE PRINCIPAL ARTICLES IN THIS VOLUME ARE AS FOLLOWS[1]:—

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[1] The Editor has to record the great loss sustained by the Profession through the lamented death of Dr. John Duncan. Among the latest subjects that engaged his attention were the articles on "Capillaries" and "Electrolysis," which he contributed to this work.

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Bowlegs. *See* DEFORMITIES.

Brachial Plexus, Surgical Affections of.

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SURGICAL ANATOMY.—Occupying the posterior triangle of the neck, and extending thence to the sub-clavicular fossa, the brachial plexus is formed by the anterior divisions of the four lower cervical nerves, the first dorsal nerve, and a part of the fourth cervical root. Its roots may be here considered together with the plexus itself, and thus defined: It lies within a triangle whose base is the spine, from the fourth cervical to the second dorsal vertebra, and whose apex is internal to the coracoid process. Its *roots*, gradually approaching one another, pass from the intervertebral foramina through and between the anterior and middle scalene muscles, and then decussate. As a rule the fifth and sixth roots, with the slip from the fourth, join to form an upper trunk; the eighth cervical and first dorsal form a lower trunk; the seventh cervical constitutes a middle trunk. Thereafter each *trunk* sends a branch to form the posterior cord; the remainder of the lower trunk forms the lower cord; the remainder of the upper and middle trunks form the upper cord. The subsequent distribution of these *cords* is described in text-books of anatomy.

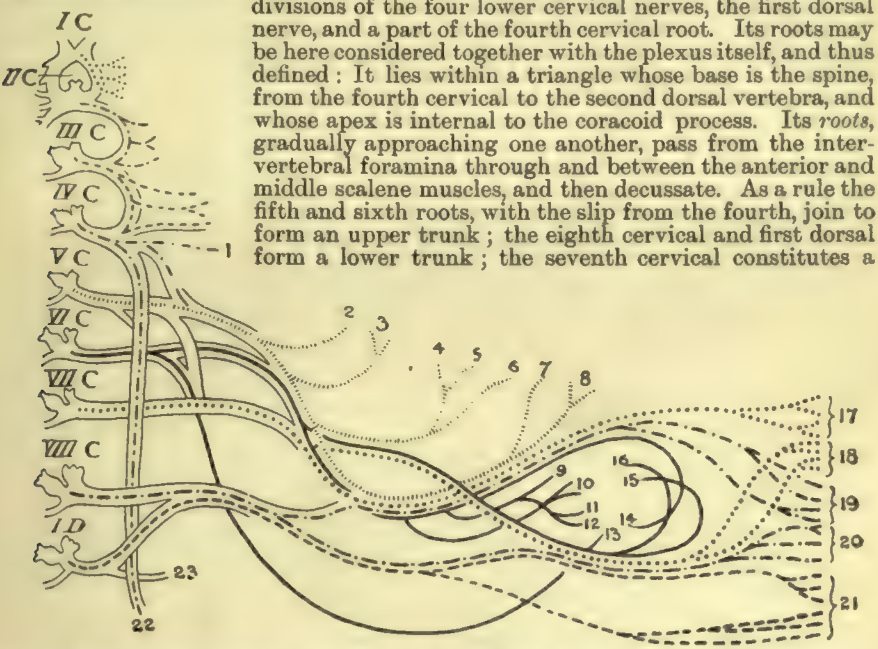

FIG. 1.—Motor distribution of cervical and first dorsal nerves (*apud* Kocher).—1. Scalene; 2. Rhomboids; 3. Supra and Infra Spinatus; 4. Coraco-brachialis; 5. Biceps; 6. Brachialis Internus; 7. Deltoid; 8. Supinator l. f. b.; 9. Subscapularis; 10. Pectorals; 11. Lat. Dorsi; 12. Teres Major; 13. Serratus Magnus; 14. Triceps; 15. Pronator quad.; 16. Pronator Teres; 17. Extensors of Wrist; 18. Flexors of Wrist; 19. Extensors of Fingers; 20. Flexors of Fingers; 21. Small Muscles of hand and digits; 22. Phrenic to diaphragm; 23. To pupil and orbit.

The attempt to trace anatomically the destination of each root of the brachial plexus is surrounded with difficulties, but the experimental studies of Ferrier and Yeo and of Sherrington, and the clinical observations of Duchenne, Erb, the writer, Head, Kocher, and others, have now made us tolerably familiar with their course. The following diagrams indicate the results of Kocher's most recent work upon this subject. In Fig. 1 the various motor roots are traced out through the plexus to their respective muscles; in Figs. 2 and 3 the sensory distribution of the same roots on the front and back of the limb are also shown; but it is probable that the outer part of the area assigned by Kocher to the first dorsal root is in reality supplied by the eighth cervical, the various "root areas" thus lying in parallel series in the order in which we may suppose the body segments to have been pushed out from the trunk by the lateral projection of the limb bud.

With the aid of the above diagrams it will generally be found possible to locate with precision the extent to which the brachial plexus is involved in any pathological process, but it is also of the greatest importance to be able to estimate how far from the cord any given lesion is situated. For this purpose we may indicate the order of origin of some of its highest branches :—

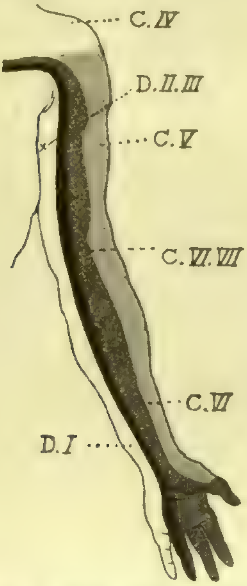

Fig. 2.—Front of upper limb showing sensory distribution of cervical and two upper dorsal nerves (*apud* Kocher).

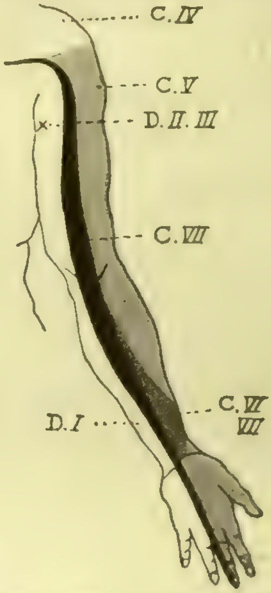

Fig. 3.—Back of upper limb showing sensory distribution of cervical and two upper dorsal nerves (*apud* Kocher).

The first branch of the first dorsal nerve passes to the cervical sympathetic and then supplies the dilator iridis and the muscle of Müller.

The fifth and adjacent roots give off branches above the plexus in the following order, as we pass downwards from the spinal cord :—

(*a*) To the rhomboids (5th).
(*b*) To the subclavius (5th and 6th).
(*c*) To the serratus magnus and supra- and infra-spinati (5th and 6th).

By noting whether the above muscles are or are not involved in a paralysis, we can determine how far from the spine is the lesion causing such paralysis.

Wounds.—The whole or a part of the brachial plexus may be divided by cuts, stabs, and other similar wounds of the neck, and especially by those of the posterior triangle or of the subclavicular fossa, although it is not exempt from injury in deep wounds of other regions of the neck. Such wounds are, however, by no means common, and it is especially rare for the entire plexus to be divided thereby. *Gunshot wounds* are hardly ever observed except in association with other injuries so extensive as to render the damage done to the plexus comparatively insignificant.

The *symptoms* are immediate motor and sensory paralysis, varying in their distribution according to the exact position of the injury, but tending especially to appear in the distribution of the upper and more exposed roots of the plexus. These symptoms will be more fully considered in the description of tears and contusions. In many cases, and especially in those of injury situated low down and beneath the clavicle, the great vessels of the upper limb will also be wounded, and their condition will most urgently demand the attention of the surgeon.

The *treatment* of these rare injuries is sufficiently obvious. An incision will often be required to enable us to deal with severe hæmorrhage; but in all cases *primary suture* of the divided nerve-trunks should be adopted, the plexus being properly exposed in its wounded area, and the divided ends of the nerves secured. We must also follow the general rule, that if primary suture has not been adopted or has failed in its object, and if septic changes have ensued, we must await the healing of the wound and the restoration of an aseptic condition before resorting to *secondary suture.*

Tears and Contusions.—It is hardly practicable to discuss separately these two classes of injury, especially as in many cases it is uncertain what the precise mechanism of that injury may have been.

Causes.—The injury is generally produced by sudden pulling of the arm upwards above the head, as in traction upon the child's arm or axilla in obstetrical operations ("obstetrical paralysis" of Duchenne), or, in adults, from the fixing of the arm in revolving belts or during the act of falling, from clutching with the hand at some outstanding object. In nearly all cases it will be found that the whole of the upper limb has thus been forcibly dragged upwards and backwards. Less commonly the injury is associated with a fracture of the clavicle due to direct violence, and often compound, the broken ends of the bone being driven down upon the subjacent nerves; with a dislocation of the shoulder causing similar conditions; or with injury to the axilla, as from pressure in the reduction of luxations.

In many cases the *mechanism* of the injury consists in the *tearing* of the plexus by a direct pull, when either the roots are avulsed from the spinal cord or some or all of them are torn across outside of the vertebral canal. In some cases the lesion is clearly a *contusion* resulting from a direct blow, or from the driving in of a broken or dislocated bone. It would also appear that not a few cases are due to crushing of the plexus between the unbroken clavicle and the first rib, against which the collar-bone has been forcibly drawn backwards (Ross).

The *symptoms* are similar in kind in all cases, but differ in extent according to the position of the lesion and the number of cords torn. In *complete* lesions they involve all the nerve-roots; in many the injury is seated at some little distance from the spine, and the highest branches (those for the eye, rhomboids, etc.), escape. In the very characteristic group of *partial* injuries described by Erb and by Duchenne the fifth or fifth and sixth roots alone suffer, possibly owing to the fact that they are readily compressed by forcible retraction of the clavicle.

In a complete case the upper limb of the affected side hangs perfectly flaccid and is absolutely anæsthetic, save over the upper deltoid region, which is supplied from the cervical plexus, and on the axillary aspect to which the second or second and third dorsal nerves send sensory fibres. At the same time we find contraction of the pupil, or rather incapacity for dilatation (best observed in a dim light), and contraction of the palpebral fissure. Pain is not usually a prominent symptom, but may be experienced in the neck or referred to the upper limb. Vasomotor and "trophic" symptoms follow as in other injuries of nerves, and arthritis of the elbow-joint is especially common as a sequela.

This clinical picture is liable to indefinite variation in cases of varying extent, locality, and severity of injury, but such details can be most readily appreciated by reference to the functions of the various nerve-roots as previously indicated. It is necessary, however, to refer to the common type of partial lesions known as *Erb's paralysis* or *Duchenne's obstetrical paralysis*, in which the fifth root is alone torn or compressed, and in which we find only paralysis of the deltoid, biceps, brachialis internus, and spinator longus muscles, with anæsthesia limited to the territory assigned in our diagram to the fifth cervical root—the anæsthesia being often very incomplete or in the slightest cases absent.

Any other root or combination of roots may be involved in a partial lesion of the plexus, the fifth and sixth suffering with a frequency next most common to that of the fifth cervical.

The *diagnosis* presents no real difficulty, but mistakes constantly arise from regarding a hysterical monoplegia as a paralysis of the brachial plexus. In the hysterical affection we do not find the anæsthesia and paralysis limited to the distribution of one or more nerve-roots; wasting of muscles is less marked than in injuries of the plexus; anæsthesia is usually more prominent than paralysis; the "stigmata of hysteria" are generally found, the most prominent being partial hemi-anæsthesia, retraction of the field of vision, and achromatopsia—all on the affected side of the body. From an injury of the cervical cord a tear of the brachial plexus differs in being absolutely unilateral, and in the absence of any symptoms involving the lower limb or the visceral reflexes. Cerebral paralyses do not present the perfect monoplegia, flaccid paralysis with anæsthesia, and orbital symptoms which characterise injury of the brachial plexus.

The *course* of the injury is variable. Many cases present little or no improvement from the moment of the accident; others recover slowly, anæsthesia disappearing before paralysis. In the majority the ultimate prognosis is very bad; but, on the other hand, recovery may occur after long intervals. Contusions are far less serious than tears, and hence we must give a worse prognosis in "complete" cases—in which the plexus is probably torn across or avulsed from the cord—than in Erb's paralysis or in fractures of the clavicle and other similar conditions.

Treatment.—The general management of symptoms and of trophic troubles does not differ from that employed in other regions, but the question arises whether, and if so when, we are justified in operating upon the injured nerves. A dogmatic answer is hardly possible, but the following rules may be found useful:—(1) Injuries of the clavicle with severe brachial symptoms certainly call for operation with reposition and wiring; tears of nerves should then be looked for and sutured. (2) Where the mechanism of injury indicates a tear with probable separation of the ends of the nerves, suture ought to be attempted at once, but is practicable only if the lesion be at some little distance from the spinal cord (as indicated by absence of pupillary symptoms or of paralysis of the rhomboids or serratus magnus). (3) In other cases we should at least defer operation for some weeks and be satisfied that recovery of function is not in progress; (4) In old-standing cases operation is at least worth a trial, although by no means hopeful.

In hopeless cases of Erb's paralysis or of obstetrical paralysis the affected limb may be rendered far more useful by means of an artificial support to the elbow to counteract the paralysis of its flexors, or by arthrodesis of that joint; and in hopeless cases of complete paralysis amputation may be useful to remove a useless limb, especially when trophic ulcerations render it a source of danger.

Pressure Lesions of the brachial plexus are most typically met with when caused by a supernumerary cervical rib, but all kinds of tumours of the neck and axilla, aneurisms of the subclavian artery, and the like, are liable to compromise its structures. An accessory cervical rib usually produces symptoms in early adult life by pressing upon the lower cervical roots, and such symptoms are not rarely bilateral. Most commonly there is first observed a neuralgia on the inner side of the forearm (first dorsal root) with or without hyperalgesia, and some loss of tactile sense passing on to distinct anæsthesia; the muscles of the hand, and to a less extent those of the forearm, become weakened and much atrophied; trophic changes may ensue in the inner fingers; the pupillary and orbital fibres escape injury, as they lie above the rib; the bony prominence is felt in the posterior triangle of the neck, and pressure on it often causes tingling pain in the inner side of the upper limb. In other cases of pressure the symptoms are similar in nature, but vary indefinitely in extent and degree. The *treatment* of all these cases consists in the removal of the source of pressure.

Neuralgia of the brachial plexus is common, and may involve the whole, or far more often a portion only, of its roots or branches. In the majority of such cases seen by the writer, distinct rheumatic lesions have also occurred, and the neuralgia has yielded to the use of salicylates; it may then be regarded as a truly rheumatic manifestation. Influenza appears to cause some cases, which are probably due to a localised neuritis. Many are due to injury, such as falls or blows upon the head, neck, or shoulder, and are presumably due to the formation of perineural cicatrices. Others result from gross pressure, and in all cases the posterior triangle of the neck and the axilla should be most carefully examined for any such source of pressure. Lastly, there remain, here as elsewhere, a group of neuralgias of which the causation and pathology are equally obscure.

The essential symptom is pain usually in the distribution of a definite nerve root or branch, often intermittent, constantly associated with tingling and stabbing, and either with or without loss of precise tactile sensation.

In addition to the general *treatment* of neuralgia, we may in severe cases adopt one of the two following operations:—(1) Stretching the plexus through a free incision has given some excellent results and is well worthy of trial. (2) Division of the posterior roots of the spinal nerves after laminectomy (Bennett, Abbé) has not yet been attempted sufficiently often to have obtained a recognised position in surgery, but is practicable and advisable in the most hopeless cases.

Operations.—Exposure of the brachial plexus may be called for under the following circumstances:—(1) For primary suture of wounds and tears; (2) for neuroplastic operation in old injuries; (3) for the removal of sources of pressure; (4) for nerve-stretching.

In many of these cases the exact nature of the procedure will be governed by collateral considerations, such as the presence of an accidental wound, a fracture, or a tumour; but in others the best incision is a vertical one along the anterior border of the trapezius. For more extensive operations this incision can be carried down the anterior border of the deltoid, the clavicle being temporarily resected when a most satisfactory field of operation is secured. After division of the platysma and deep cervical fascia, and ligature or retraction of numerous veins, the plexus is at once seen in the floor of the wound escaping from the outer border of the anterior scalene muscle, and covered by a lax cellular tissue which is readily torn apart. The lowest root and cord are overlaid by the subclavian and axillary arteries, but a little care is alone needed for its identification and separation. After thus exposing the nerves, suture or stretching is readily performed by ordinary methods.

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Brain.—This subject will be here considered in the following articles:—

1. **Physiology of.**
2. **Diseases of Blood-vessels.**
3. **Cerebral Tumours.**
4. **Atrophy, Hypertrophy, Cysts, etc.**
5. **Intracranial Surgery.**

Brain, Physiology of.

ANATOMICAL 6
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THE CEREBELLUM AND ITS PEDUNCLES 15
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THE outer surface of each hemisphere is divided into four lobes by three main fissures—those of Sylvius and Rolando and the parieto-occipital fissure.

The fissure of Sylvius has two limbs, a short vertical limb, sometimes subdivided into two, and a long horizontal limb, which passes obliquely backwards and somewhat upwards, and at its hinder termination bends upwards for a short distance at an obtuse angle to the main stem so as to be directed towards the great longitudinal fissure.

The fissure of Rolando is from two and a half to three inches long. Its upper extremity begins near the great longitudinal fissure, and extends downwards and forwards, forming an angle of 67·5° with that fissure. It does not quite reach the Sylvian fissure. Its course is somewhat sinuous, having a double forward convexity.

The parieto-occipital fissure is seen midway between the upper end of that of Rolando and the tip of the occipital lobe. It extends for only a very short distance on the outer surface of the hemisphere.

The frontal lobe is bounded by the fissures of Sylvius and Rolando; the parietal lies between the fissures of Sylvius, Rolando, and the downward continuation of the parieto-occipital fissure to meet the backward imaginary prolongation of the Sylvian fissure; the occipital lobe lies behind the continuation of the parieto-occipital fissure; the temporal lies below the Sylvian fissure, and is bounded behind by the occipital lobe.

In the frontal lobe there are three horizontal convolutions, which are termed respectively *superior*, *middle*, and *inferior*, or *first*, *second*, and *third*, and one vertical, the *ascending frontal* or *precentral convolution*. This is partly bounded in front by the precentral sulcus, which, however, is interrupted so as to allow part of the hinder termination of the three horizontal convolutions to become continuous with the ascending frontal.

The parietal lobe is divided into an upper and lower portion by the long curved *interparietal fissure*. Above this lie the *ascending parietal* and *superior parietal* gyri. Of these two, which are continuous with each other, the former lies along the posterior margin of the fissure of Rolando, the latter along the border of the great longitudinal fissure as far back as the parieto-occipital sulcus. Below the interparietal fissure lie the *supra-marginal* and *angular* gyri. The former of these is situated above the hinder portion of the horizontal limb of the Sylvian fissure. It is arbitrarily divided from the angular gyrus by an imaginary line prolonged from the upward bend of the Sylvian fissure to the interparietal sulcus. The angular gyrus lies between this line, the posterior limb of the parietal lobe as above defined, and a line prolonging backwards the Sylvian fissure. The supra-marginal is frequently described as passing round the upturned extremity of the fissure of Sylvius to become continuous with the superior temporal convolutions and the angular gyrus. The angular gyrus, in this case, is said to bend round the hinder end of the parallel sulcus to terminate in the second and third temporal gyri.

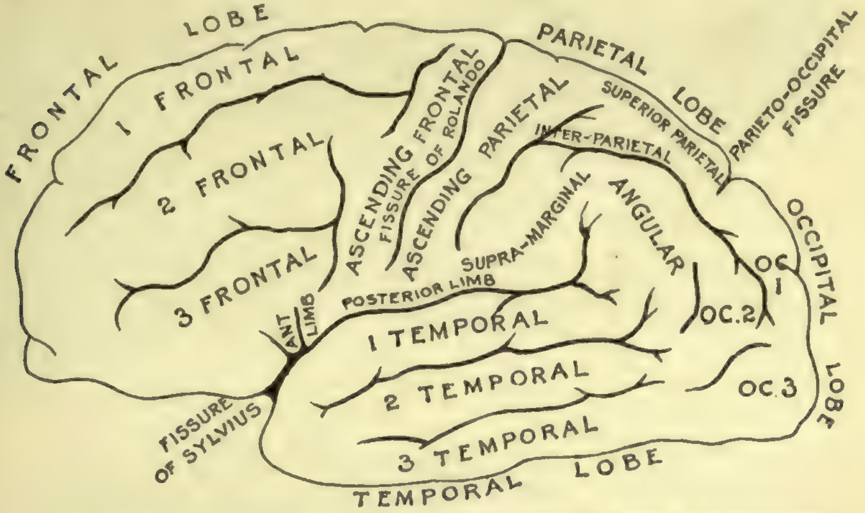

FIG. 1.—Outer surface of left cerebral hemisphere, showing the lobes, fissures, and convolutions.

In the temporal lobe an *upper*, *middle*, and *lower*, or *first*, *second*, and *third* temporal convolution are recognised. These are separated from each other by two sulci more or less parallel to the Sylvian fissure, of which the upper is generally termed the "parallel sulcus."

In the occipital lobe three convolutions, *first*, *second*, and *third*, are generally described. These are not well delimited from each other, and are connected with the adjacent convolutions by annectant gyri.

An additional lobe, the *island of Reil*, can be seen if the sides of the fissure of Sylvius are held apart. On it are four or five radiating convolutions.

On the median aspect of the hemisphere three important sulci are seen. The *calloso-marginal* sulcus begins below the rostrum of the corpus callosum, and follows the curve of that structure at a distance rather less than half-way from the free margin of the hemisphere. When almost opposite the splenium of the corpus callosum it suddenly bends backward almost at a right angle, and reaches the border of the hemisphere a little behind the fissure of Rolando. The *parieto-occipital fissure* passes downwards and forwards and ends a little below the splenium of the corpus callosum. It is joined near the middle of its length by the *calcarine fissure,* which is directed from this point towards the tip of the occipital lobe.

The convolutions of importance on this surface are the *marginal* above, and the *gyrus fornicatus* below the calloso-marginal sulcus. Between the upturned part of this fissure and the parieto-occipital sulcus is the square *quadrate* lobe, or *precuneus.* That portion of the marginal gyrus which is opposite to the upper ends of the ascending frontal and parietal convolutions is often termed the *paracentral* lobule. Between the parieto-occipital and the calcarine fissures lies the *cuneus.*

On the inner surface of the temporal lobe are the *uncinate,* near its anterior tip; the *lingual* and *fusiform* gyri below the calcarine fissure; and the *inferior occipito-temporal,* a long gyrus extending, as its name indicates, along the inferior margin of both occipital and temporal lobes.

Blood-supply.—The arterial supply of the brain is derived from the two internal carotid and the two vertebral arteries. The vertebrals enter the cranial cavity through the foramen magnum, and at the lower margin of the pons they unite to form the basilar artery, which divides opposite the beginning of the interpeduncular space into the two posterior cerebral arteries. Each internal carotid divides, on entering the cranium, into the anterior and middle cerebral arteries. The two anterior cerebral arteries are connected, close to their origin, by the anterior communicating artery; the middle and posterior cerebrals on each side are united by the posterior communicating artery. There is thus established an arterial intercommunication (termed the "circle of Willis") of the greatest importance for the maintenance of an efficient supply to the hemispheres, which can be carried on even if one of the main trunks becomes occluded.

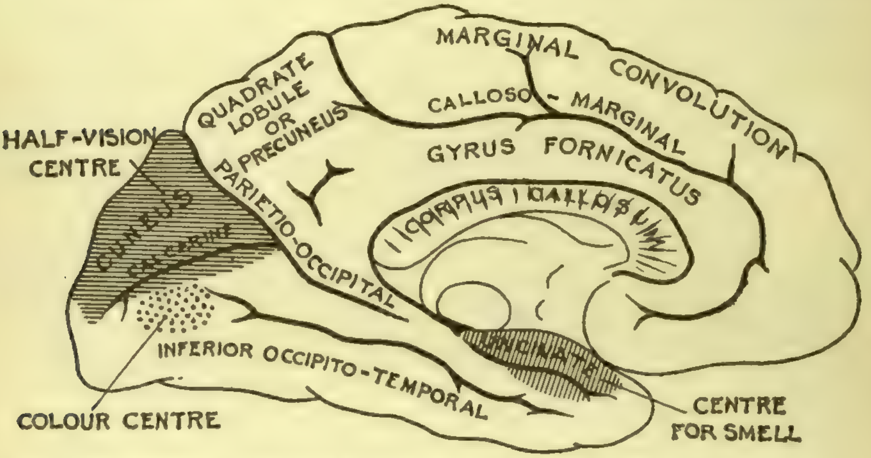

Fig. 2.—Median aspect of left cerebral hemisphere, showing the fissures and main convolutions, also the centres for smell, for vision, and colour vision.

The three cerebral arteries, anterior, middle, and posterior, supply each hemisphere, giving off branches to the cortex and the immediately subjacent white matter, on the one hand (cortical branches), and to the central ganglia and the superjacent centrum ovale, on the other (central branches). It is important to remember that there is no anastomosis between the cortical and the central groups of vessels, so that the results of occlusion of one group of vessels are independent of those of the other. Again, the anastomosis between the main and the subsidiary branches of the cerebral arteries is so imperfect that for practical purposes they may be regarded as being, though they are not in reality (Heubner), terminal arteries. Consequently occlusion of one artery or one of its main branches leads to a softening strictly limited to the area supplied by the affected vessel.

Again, the smaller twigs which enter the cerebral cortex, and the central arteries to the basal ganglia, are devoid of all anastomosis with each other, and are true terminal arteries. The cortical branches are found in the subarachnoid space, and before entering the cortex they give off branches at right angles which may, according to Duret, be divided into two groups —a short *cortical* group, which for the most part terminates in a fine capillary network in relation to the large pyramidal and ganglion celled layer of the gray matter, and a longer *medullary* group, which, after passing right through the gray matter, supplies the underlying white substance for a depth of a half to three-quarters of an inch. As already said, this latter group does not anastomose with the central branches, so that between the territories supplied by these two groups of vessels there is an area so feebly nourished that it is apt to be the seat of lacunar softening in old people.

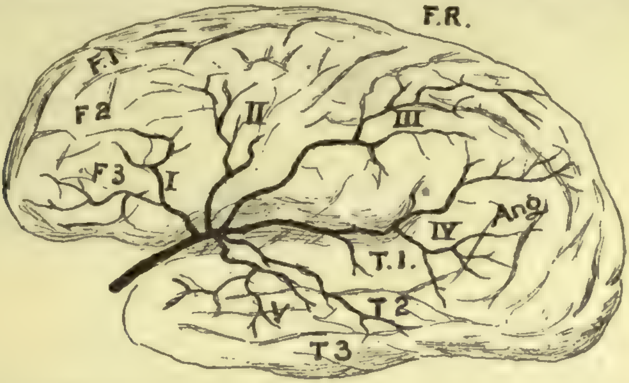

Fig. 3.—To show the distribution of the five branches of the middle cerebral artery.

The *anterior cerebral* is distributed on the inner aspect of the hemisphere as far back as the parieto-occipital fissure, to the orbital surface of the frontal lobe, to the anterior two-thirds of the second frontal, and to the whole of the superior and the upper half of the ascending frontal convolutions. Its central branches supply the upper part of the head of the caudate nucleus and are occasionally the seat of hæmorrhage.

The *middle cerebral* is the largest as well as the most important of the cerebral arteries, having regard to the areas of the cortex which it supplies and the frequency with which, more especially on the left side, it is affected by disease. Its five cortical branches are distributed to—(1) the third frontal convolution; (2) to the lower two-thirds of the ascending frontal, and the hinder end of the second frontal; (3) to the ascending parietal, superior parietal, and supra-marginal convolutions; (4) to the angular gyrus, the supra-marginal, and the posterior end of the first and second temporal convolutions; (5) to the anterior part of the first temporal and the anterior two-thirds of the second temporal convolutions.

The central branches, which leave the main stem near its origin, enter the anterior perforated space and constitute the main supply of the basal ganglia. They pass upwards at first through or external to the lenticular nucleus, and are then directed inwards through the internal capsule either to the caudate nucleus or to the optic thalamus, being termed respectively *lenticulo-striate* and *lenticulo-optic* arteries. One of the former group which ramifies at first on the outer surface of the lenticular nucleus is so frequently ruptured that it has been termed by Charcot the *artery of cerebral hæmorrhage.*

The *posterior cerebral* supplies the remainder of the cortex, namely, the inner aspect of the occipital and temporal lobes (including the cuneus, the lingual, fusiform, and uncinate gyri), the outer occipital and the inferior temporal convolutions.

Its central branches supply the posterior part of the optic thalamus, the crus, the nucleus of the third nerve, and the external geniculate body.

The medulla, pons, and cerebellum derive their supply from the basilar and the two vertebral arteries. Those to the medulla and pons are divided into *medial* and *lateral* or *radicular* branches. The medial arteries are the chief supply of the nuclei (*bulbar* to the hypoglossal and spinal accessory; *subpontine*, from the basilar, for the vagus, glossopharyngeal and auditory nuclei; *midpontine* and *suprapontine*, for the nuclei of the remaining cranial nerves below the third). The *lateral* branches, as their other name implies, supply the nerve-roots. They divide into ascending and descending branches—the former passing with the nerve-roots into the medulla and supplying the nuclei of the nerves they accompany, the latter passing outwards with the nerve.

The *cerebellar arteries* are the *superior*, *middle*, and *inferior*. The first named are derived from the basilar, and supply the middle lobe, and the upper part of the hemispheres of the cerebellum. One branch to the nucleus dentatus (arteria corporis dentati of Duret) merits special mention as being the most frequent source of cerebellar hæmorrhage. The middle and inferior cerebellar arteries are derived from the vertebrals, and between them supply the remainder of the cerebellum.

Venous Circulation.—The *venous blood* is collected from the cerebrum mainly by two channels, the superior longitudinal and the straight sinuses. The former, which runs from before backwards along the upper margin of the falx cerebri, drains the greater portion of the outer and inner surface of the hemispheres. The cortical veins, some ten or twelve in number, enter the sinus in an anterior direction—an arrangement supposed to be designed for the too rapid removal of venous blood from the cerebrum, but more probably merely the result of the growth of the cerebrum in a forward direction. The straight sinus is formed by the union of the inferior longitudinal veins and the vein (or veins) of Galen. The former runs in the inferior margin of the falx, from which, as well as, to some extent, from the inner surface of the hemispheres, it derives its blood. The veins of Galen which lie near each other between the layers of the velum interpositum are formed by the union of veins from the caudate nucleus and optic thalamus and also from the choroid plexus in the lateral and third ventricles. As the veins of Galen pass over the pineal gland they are apt to be compressed by tumours of that structure, thus giving rise to dropsy of the cerebral ventricles (secondary hydrocephalus). The straight and superior longitudinal sinuses unite at the torcular Herophili (opposite the internal occipital crest) to form the lateral sinus. This occupies the well-known groove in the occipital bone and the postero-inferior angle of the parietal bone, lying in this part of its course in the attached margin of the tentorium cerebelli. Then it grooves the mastoid part of the temporal bone and enters the jugular foramen. One lateral sinus is usually larger than its fellow. The relations of the sinus to the temporal bone are of the utmost importance to the surgeon (see p. 73).

The cavernous sinus at the side of the body of the sphenoid bone receives the ophthalmic vein, communicates with its fellow by the circular sinus round the pituitary body, and delivers its blood to the lateral sinuses through the superior and inferior petrosal sinuses. The former of these passes along the groove in the upper margin of the petrous bone and in the attached margin of the tentorium cerebelli, and enters the lateral sinus in the groove in the mastoid-temporal bone. It receives some branches from the upper surface of the cerebellum, the inferior surface of the cerebrum, and from the tympanum. The inferior petrosal joins the jugular vein at the jugular foramen, passing to this point along the junction of the basi-occipital and petrous-temporal bones. It receives blood from the under surface of the cerebellum and from the internal ear. The occipital sinus, sometimes single, sometimes double, in the falx cerebelli, joins the posterior spinal veins and ends in the lateral sinus.

Special mention must be made of the connections of the vessels in the dura mater with veins entering it either from the cranial bones or from structures outside the skull, these vessels being of importance as conveying infective processes from without and leading to sinus thrombosis. The *ophthalmic veins* from the eye and orbit have been already mentioned as entering the cavernous sinus through the sphenoidal fissure. The *mastoid emissary vein* passes from the lateral sinus through the mastoid foramen. The *parietal emissary* vein connects the superior longitudinal sinus with the veins of the scalp. There are others of less importance which do not require mention.

LYMPHATIC CIRCULATION OF THE BRAIN.—The researches of Obersteiner, His, Bevan Lewis, Retzius, Leonard Hill, and others have elucidated the lymphatic paths and the conditions of the secretion of lymph and of its absorption. In the gray matter of the cortex each nerve lies in a small sac, lined with endothelium—the *pericellular lymph-sac.* This is continuous with a *perivascular lymph-channel,* which surrounds the vessels (arteries, veins, and capillaries) within the gray matter. These channels open into the reticulated subarachnoid space, which, in the fissure of Sylvius and at the base of the brain, form larger spaces, termed *lymph-cisterns,* which may under certain pathological conditions become over distended with fluid, but which in the normal brain are, according to Hill, little more than potential spaces, capable of containing more fluid if the amount of blood in the cranial cavity is diminished. In addition to this canalicular system, Bevan Lewis thinks that the neuroglia cells of Deiters serve as absorbents of lymph. He bases his opinion on the fact that the protoplasm of these cells is of a semi-fluid nature and that the cells send one process to join the adventitial sheath of the vessels. He supposes that the cells absorb fluid from the brain-substance and deliver it by their long process to the perivascular lymphatics. These cells he terms the *lymph connective system.*

The cerebro-spinal fluid within the ventricles is mainly secreted by their choroid plexuses. Under ordinary circumstances the fluid passes from the ventricles of the cerebrum through the aqueduct of Sylvius to the fourth ventricle. From this the fluid escapes through the foramen of Majendie in the roof of the ventricle into the subarachnoid space. From the subarachnoid space the exit for the cerebro-spinal fluid is by way of the lymphatics of the nerve-roots and those surrounding the larger blood-vessels of the brain and cord. Further points of clinical importance regarding the cerebro-spinal fluid will be found under the section " Nose, Diseases of."

FUNCTIONS OF THE CEREBRAL CORTEX. — The clinical researches of

Hughlings Jackson, followed by the experiments of Fritsch and Hitzig, Ferrier, Horsley, Beevor, and others, have shown that there are certain areas in the cortex, stimulation of which produces movements on the opposite side of the body, and removal of which is followed by paralysis of the same movements. These areas are located on both sides of the fissure of Rolando, mainly in the ascending parietal and frontal convolutions and their terminations in the median aspect of the hemisphere, viz. the paracentral lobe; also to some extent in the superior parietal, and in the posterior ends of the first, second, and third frontal convolutions. These areas, termed "motor areas," have been marked out with great precision and minuteness by Horsley and Beevor, and have been found by the former to correspond closely with those indicated by disease in man. Their position is shown in the accompanying diagram. It may be stated generally that the centres for the leg are in the upper part of the motor area, those for the arm in the intermediate portion, and those for the mouth, the tongue, lips, and larynx in the lowest part. The movements of the lower extremity are arranged along the margin of the great longitudinal fissure *from before backwards*, as follows: hip, ankle, knee, great toe, and small toes. The movements of the arm are arranged *from above downwards*, as follows: shoulder, elbow, wrist, fingers, index, thumb. The movements of lips, mouth, larynx, and pharynx are all represented on the ascending frontal and parietal convolutions below the lowest anterior curve of the fissure of Rolando. Their relative position is indicated in the diagram, and need not be specially described.

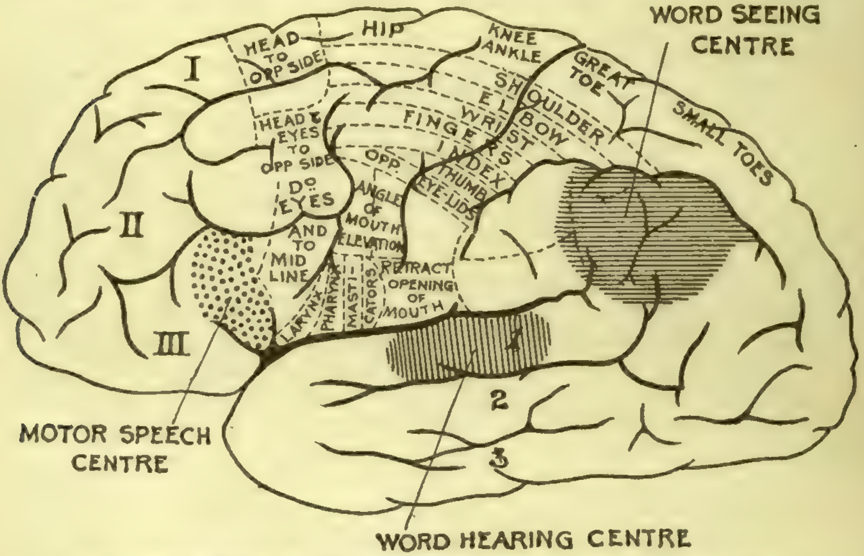

FIG. 4.—Outer surface of left hemisphere indicating the "motor" and "sensory" areas.

Additional areas for the movements of the head and eyes to the opposite side are figured at the hinder extremities of the two upper frontal gyri. The lower frontal gyrus, in the left side, often termed Broca's convolutions, is the motor speech-centre (see "Aphasia").

The positions within the internal capsule of the conducting strands to and from the various areas of the cortex are of importance to the clinician. Speaking generally, the motor fibres occupy the "knee" and the anterior two-thirds of the posterior limb of the capsule, being therein distributed from before backwards, eyes, head, mouth, tongue, upper limb, trunk, lower limb. The position of the fibres subserving various functions is indicated in the accompanying figure. The anterior limb of the capsules conveys fibres from the non-excitable remainder of the frontal lobe to the cells in the pons. The formation of these fibres is as yet unknown. The posterior third of the hinder limb of the capsule contains the ingoing sensory fibres on their way to their termini in the cortex, and also descending fibres of unknown function from the temporal lobe to the pons.

Within the cerebral peduncle they are arranged from within outwards, as follows: arm, face, leg.

Sensory Centres.—These are not determined with the same degree of definiteness as the motor areas, owing, no doubt, to the greater difficulty of interpreting the results of experiments in animals.

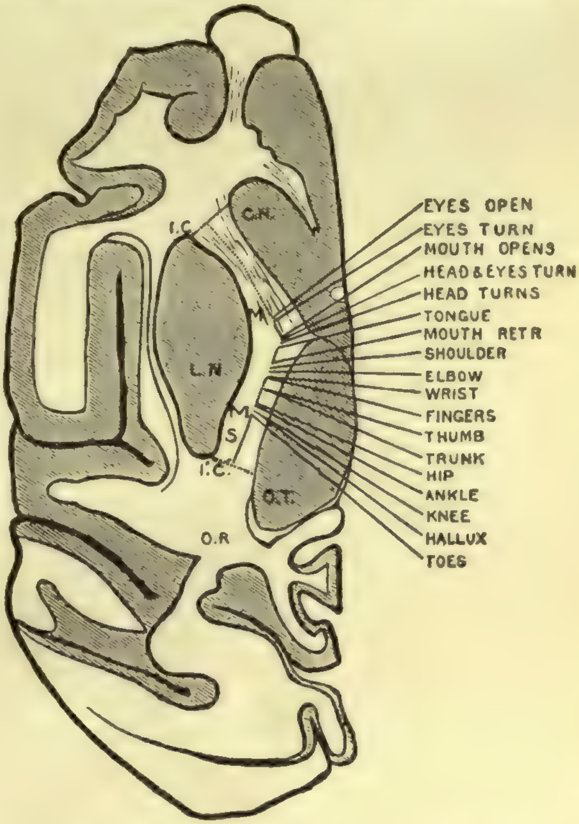

Fig. 5.—Horizontal section of left hemisphere of the brain of a monkey to show the excitable area of the internal capsule; the movements of the different parts of the body, and the order in which they were obtained when the fibres were stimulated electrically, are shown in the margin. C.N., Caudate nucleus, and L.N., Lenticular nucleus of the corpus striatum; O.T., Optic thalamus; I.C., Internal capsule; M., Excitable or motor part; S., Sensory part; O.R., Optic radiations.—C. E. Beevor, *Diseases of Nervous System.*

Sight.—The centre for sight seems to be the most definitely ascertained, and both experiment and clinical observations indicate that in the neighbourhood of the calcarine fissure, especially in the cuneus and in the lingual and fusiform lobes, lies the "half-vision" centre. If this region is destroyed, blindness results in the opposite halves of the fields of vision of the two retinæ (homonymous hemianopia). It has recently been rendered probable by Mackay and Dunlop that the "half colour-vision" centre is situated in the fusiform lobe (Fig. 2).

Hearing.—The centre for hearing is in the middle of the first temporal convolution. The limited number of clinical observations available which have been confirmed by post-mortem examination seem to indicate that destruction of the upper temporal convolutions produces deafness in the opposite ear, but that this is soon compensated for—a result easily comprehensible when the relations of the central paths from the auditory nerve are taken into consideration. In the section on the auditory nerve it is shown that the cochlear nucleus sends fibres to the posterior corpus quadrigeminum of its own side as well as to the opposite side, and as this ganglion is connected with the internal geniculate body and through this with the auditory cortex, compensation can be easily effected.

Smell.—The centre for smell appears to be situated in the tip of the uncinate gyrus on the same, and probably, too, on the opposite, side. In disease of this region epileptic fits marked by olfactory auras have been noted.

Touch and muscular sense—The centre for the perception of tactile impressions is by some located in the gyrus fornicatus, by others in the "motor" cortex. It is certain that portions of the motor cortex may be removed without loss of tactile sensibility; but a certain loss of muscular sense in the limb supplied by the removed area has been found by Horsley, though not apparently by others. Embolic destruction of the motor area does not seem to cause any sensory defect. The sensory path to the cortex passes through the hinder third of the posterior limb of the internal capsule, and radiates outward from there towards the various cortical areas. In particular the central optic path lies in the lowest part of the optic radiation of Gratiolet. Within the pons and cerebral peduncles the sensory path is less easy to trace, but it appears to lie in the tegment somewhat internal and posterior to the gelatinous substance of Rolando, where that exists.

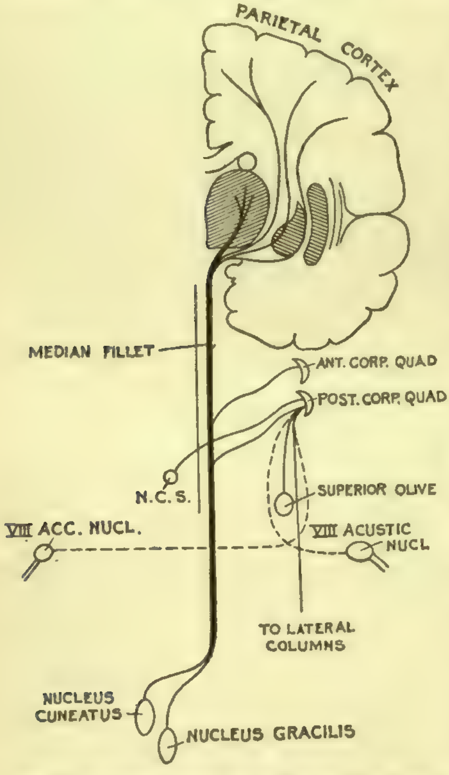

Fig. 6.—Scheme showing the distribution of the fillet.

The centripetal path for muscular sense impressions is in all probability constituted by the fillet or lemniscus. This structure arises in the nuclei of the two divisions of the posterior columns of the cord, and passes upwards along the opposite side of the raphé of the medulla, receiving, as it goes, fibres from the sensory nuclei of the fifth, eighth, ninth, and tenth nerves. The fibres of the fillet terminate in the anterior and posterior corpora quadrigemina, in the optic thalamus, and to some extent in the motor cortex.

Word-seeing Centre.—It is found that when the angular and supramarginal gyri are destroyed on the left side the power of perceiving words as words is lost. Although vision is preserved intact, words read soon have no more meaning than those of an unknown foreign language. Such a loss of perception is termed *word-blindness.* It would appear that in the above-named gyri the visual impressions of letters and words undergo a higher elaboration and become associated with definite sounds and meanings.

Word-hearing Centre.—A similar centre for the elaboration of heard sounds into words is found in the posterior part of the first temporal convolutions. Destruction of this area on the left side, while affecting hearing only temporarily on the opposite side, causes loss of understanding of spoken speech (word-deafness). The question of word-seeing and word-hearing are more fully considered in the article "Aphasia."

The anterior part of the frontal lobe is generally regarded as being associated with the higher intellectual faculties.

The functions of the caudate and lenticular nucleus and optic thalamus are not yet known. Lesions in their substance do not produce characteristic symptoms unless they press on such adjacent structures as the internal capsule or optic tract.

THE CEREBELLUM AND ITS PEDUNCLES.—The cerebellum has five lobes—a median (or vermiform), two lateral lobes, and two flocculi. The subdivisions of these will be found in any anatomical text-book, and it is unnecessary to detail them here. It may be said, however, that affections of the middle lobe and of the flocculus are apt to be accompanied by the disturbances of equilibration and of co-ordination of the movements of the extremities and eyeballs which are regarded as specially characteristic of cerebellar disease, while lesions of the lateral lobes are not infrequently latent. The reasons for this are probably to be found in the connections of the cerebellum with the brain medulla and cord.

The *inferior peduncle* of the cerebellum, or the restiform body, connects it with the medulla and cord. It is composed of (1) the direct cerebellar tract; (2) the arcuate fibres derived from the nuclei of the posterior columns of both sides of the cord, (3) from the inferior olive, (4) and from a nucleus at the side of the medulla termed the lateral nucleus. All these fibres, with the exception of those from the inferior olive, conduct in a centripetal direction and terminate in the upper part of the middle lobe of the cerebellum, mainly, but not entirely, on the same side of the mesial plane. These fibres, moreover, almost certainly conduct muscular sense impulses (using that term in its most general sense). The fibres connecting the olive with the cerebellum seem to conduct centrifugally from the latter.

The ascending antero-lateral tract of Gowers, although it does not belong to the restiform body, also enters the upper part of the middle lobe of the cerebellum. It passes upwards through the lateral part of the tegment of the pons, hooks round the fifth nerve at its emergence, and then curves round the superior cerebellar peduncle just below the posterior corpus quadrigeminum, and bends downwards into the lingual lobe of the vermis.

From the cortex of the vermis, sagittal fibres pass to two rounded nuclei in the anterior part of the lobe, termed, from their relation to the fourth ventricle, the roof nuclei. Each roof nucleus, again, gives origin to an important tract, the cerebello-vestibular tract, which Ferrier and Turner have shown to degenerate, and therefore to conduct towards the nucleus of Deiters (vestibular nucleus). This tract follows the lateral wall of the fourth ventricle partly internal and partly external to the superior cerebellar peduncle.

The cortex of the middle lobe is probably also connected with the nucleus dentatus and known as the superior cerebellar peduncle.

The convoluted structure, the nucleus dentatus, which lies at the side of the fourth ventricle on the confines of the middle and lateral lobes, also receives fibres from the cortex of the lateral lobes. These fibres probably form the majority of those which arise from the Purkinje's cells. The nucleus sends fibres to the inferior olive of the opposite side, and also through the superior cerebellar peduncle to the red nucleus and optic thalamus of the opposite side.

The roof nucleus and the nucleus dentatus (with its accessory nucleus globosus and emboliformis) are thus concerned in receiving impressions from the cortex of the middle and lateral lobes, and in giving off centrifugal fibres to the nucleus of Deiters, the inferior olive, the red nucleus, and the optic thalamus. The distribution of the fibres from the nucleus of Deiters is considered in relation to the auditory nerve, and is of special importance as regards the movements required for the maintenance of the equilibrium.

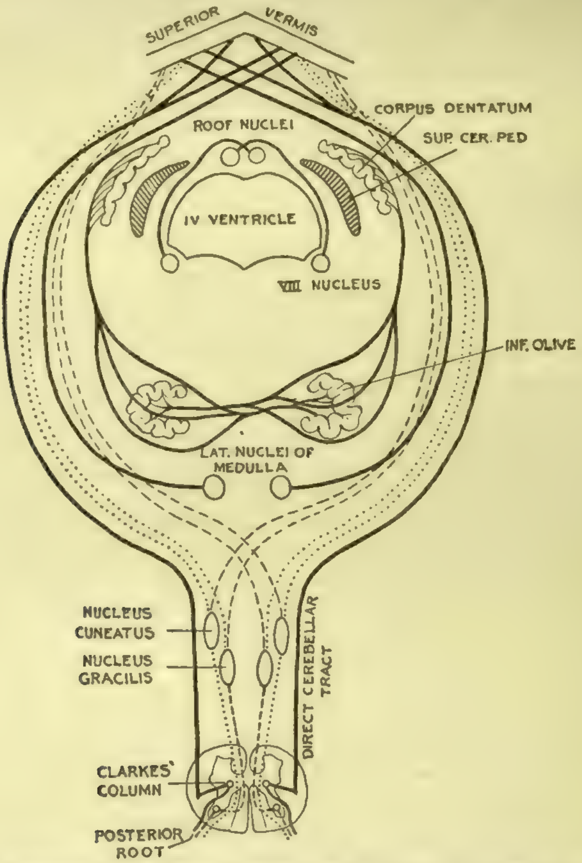

Fig. 7.—Diagram of the constituents of the inferior peduncle of the cerebellum, indicating the connection of those derived from the spinal cord with the middle or vermiform lobe of the cerebellum, and the connection of the inferior olive with the nucleus dentatus.

The *middle peduncle* of the cerebellum is formed by the superficial, middle, and deep fibres of the pons, and arises mainly from the groups of cells which lie among these fibres (the nuclei pontis). These cells are also in connection with the fibres from the frontal, temporal, and, to some extent, the parietal lobes of the cerebrum, which either end themselves or send their collaterals among the cells. The fibres from these cells pass to the cortex of the lateral lobe of the cerebellum. (A few fibres of this peduncle pass towards the nucleus pontis of the same side, and thus enter the tegment.)

It will be seen that the tracts which are connected mainly with equilibration either enter the middle lobe, or arise from its roof nuclei or the adjacent dentate nuclei; that they leave the cerebellum by the inferior and superior peduncles and by the cerebello-vestibular tract. The lateral lobes, on the other hand, which may be largely destroyed without disturbance of equilibration, receive their afferent fibres mainly from the pontine cells which are related to descending fronto-pontine and temporo-pontine tracts. The function of these tracts is not yet known.

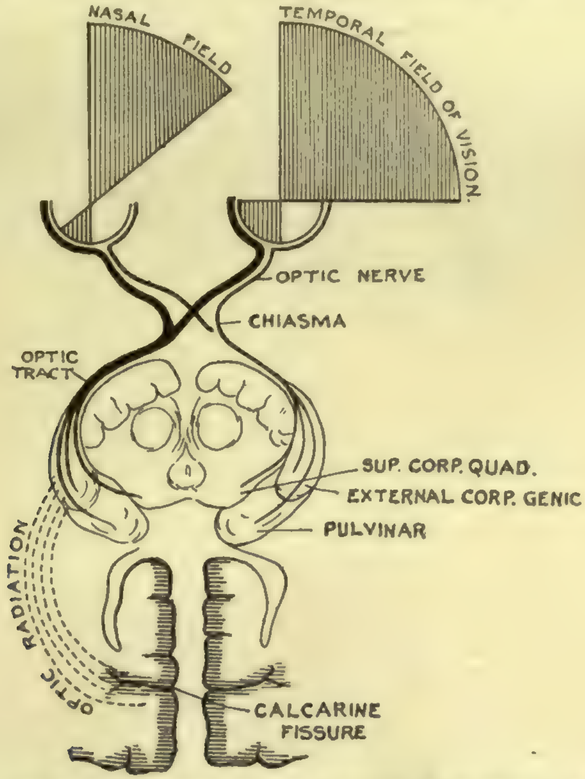

FIG. 8.—Diagram of the optic nerve, showing the relation of the cortical half-vision centre in the occipital lobe, with the corresponding halves of both retinæ and the opposite halves of the field of vision.

THE CRANIAL NERVES

The Olfactory Bulb and Lobe.—The olfactory bulb and lobe lie in the olfactory sulcus on the under surface of the frontal lobe. Near the commencement of the fissure of Sylvius the lobe divides into two roots, the outer of which crosses the fissure and ends in the tip of the uncinate gyrus of the same side, while the other root passes by way of the anterior gyrus, probably through the posterior limb of the internal capsule to the corresponding convolutions of the opposite side. Lesions of the sensory portion of the internal capsule produce, along with other forms of crossed anæsthesia, a loss of smell on the opposite side.

The Optic Nerve.—The fibres of the optic nerve are derived from the cells of the retina in such a way that those from the outer half of the retina and macula lie in the outer half of the nerve, and those from the inner half of the retina and macula occupy its inner half. At the chiasma there is a semi-decussation of the nerves, the outer halves being uncrossed and each entering the tract of its own side, while the inner halves cross into the opposite tract. The result of this process is that the fibres from the right half of each retina form the right optic tract, and the fibres from the left half of each retina form the left optic tract. Each tract passes round the crus cerebri, and terminates in a threefold manner. One group of fibres enters the external geniculate body, a second the anterior corpus quadrigeminum, and the third the pulvinar of the optic thalamus. From the geniculate body fibres pass through the optic radiation of Gratiolet to end in the cuneus, or according to Henschen in the sides of the calcarine fissure; according to Dunlop and Mackay, the centre for colour-vision is also in the fusiform lobe. This path is the central half-vision path. The fibres to the anterior corpus quadrigeminum subserve the light reflex; they terminate partly in the cells of this ganglion, and partly in relation to the centre in the third nucleus for the sphincter of the iris. (In this connection it is to be noted that the two sphincter centres must be directly connected—Weir Mitchell, Bernheimer.) The special function of the fibres to the pulvinar is not yet determined. According to Henschen they do not form part of the visual path. From the corpus quadrigeminum and pulvinar there is also a connection through the optic radiation with the half-vision centre at the side of the calcarine fissure; but the special value of these tracts is as yet unknown.

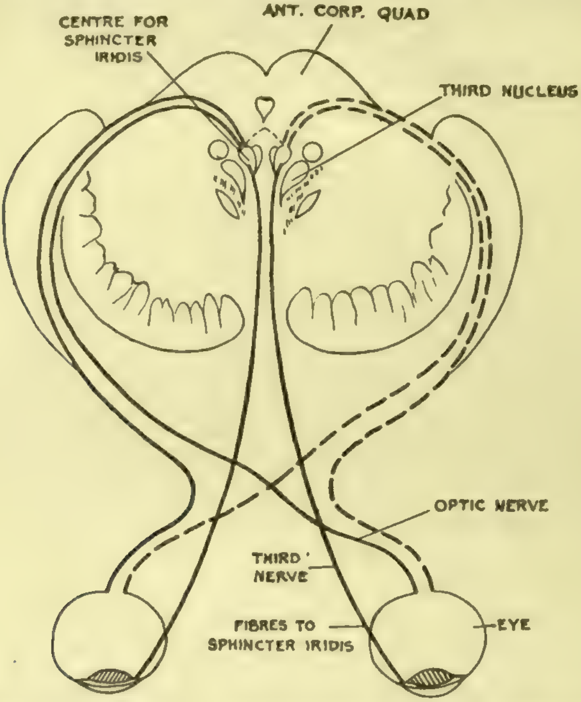

FIG. 9.—Scheme to show the course of the fibres which subserve the light reflex. Note their partial decussation at the chiasma, and their termination near the nucleus.

The Third Nerve.—The nucleus of the third or oculomotor nerve is situated in the anterior part of the gray matter surrounding the aqueduct of Sylvius, and immediately dorsal to the posterior longitudinal fasciculus. Its upper extremity is at the lower limit of the third ventricle; and its lower lies midway between the anterior and posterior corpora quadrigemina where it is almost continuous with the fourth nucleus.

The third nucleus is distinctly segmented, and each segment has probably a special function, although this is as yet only partly determined. The divisions of the nucleus are as follow:—1. A median nucleus. 2. An anterior nucleus. 3. A posterior and postero-lateral nucleus. 4. An external nucleus. 5. A postero-internal nucleus. Their relative positions are shown in Fig. 10.

The root-fibres pass through the posterior longitudinal fasciculus in separate slightly curved bundles, and pass forwards to emerge in the oculomotor sulcus on the inner side of the crus cerebri. Most of these roots arise in the nucleus of the same side; a few, mainly derived from the lower part of the anterior group, are crossed. The innermost fibres of the root arise in the median and postero-internal nuclei, and are of finer calibre than the others. They pass on the inner side of the red nucleus. The outermost fibres pass through the posterior longitudinal fasciculus and below the red nucleus. Anatomical, physiological, and clinical investigations appear to indicate that the median nucleus is the centre for convergence; the postero-internal nucleus (which seems capable of subdivision into two parts) subserves accommodation and pupil-contraction; the postero-lateral and external group supply the elevators of the globe and upper lids, viz.: the superior rectus, inferior oblique, elevator palpebræ superiors; while the anterior group sends fibres to the internal rectus and to the inferior rectus.

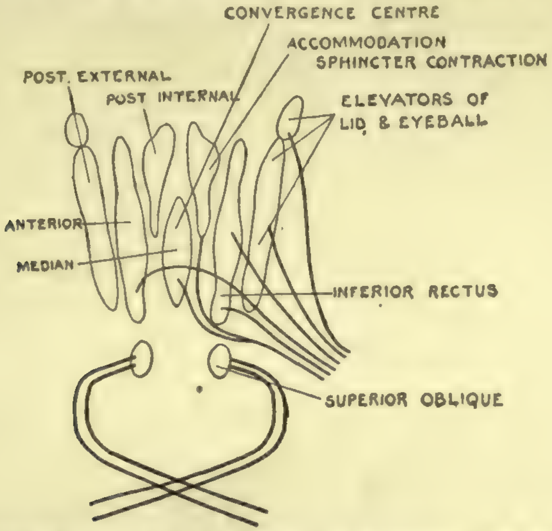

Fig. 10.—Diagram of the segments of the third nerve and their functions. The diagram shows also the position of the fourth nucleus and the course of its nerve.

The Fourth Nerve.—The nucleus of this nerve lies in the posterior aspect of the posterior longitudinal fasciculus, and is a compact ovoid group of cells almost continuous with the lower end of the third nucleus. Its fibres pass first outwards as far as the inner aspect of the upper root of the fifth nerve; they then bend downwards, and below the posterior corpora quadrigeminum in the valve of Vieussens they decussate with the roots of the other nucleus and emerge on the opposite side. The nucleus and root both appear to receive fibres from the longitudinal fasciculus, which no doubt subserve some associated movement.

The Sixth Nerve arises from a rounded nucleus which underlies the eminentia teres in the floor of the fourth ventricle, close to the median groove and immediately above the striæ acusticæ. The nucleus lies on the posterior longitudinal bundle, and has on its inferior, inner, and upper aspects the fibres of the knee of the facial nerve. Its root-fibres pass nearly directly forwards, pierce the pyramidal fibres, and emerge below the lower border of the pons in the groove at the outer side of the anterior pyramid. The nucleus also sends fibres by way of the posterior longitudinal fasciculus to the internal rectus muscle of the opposite side; and in this way it serves as the nucleus for the conjugate deviation of the eyeballs to its own side. It has also important connections with the cochlear and vestibular nuclei of the auditory nerve, which, no doubt, subserve important reflex acts. With the vestibular nuclei (nucleus of Deiters and dorsal acustic nucleus) it is connected directly by fibres passing inwards from the latter. With the cochlear nucleus it is united through the corpus trapezoideum, the superior olive, and a bundle of fibres from the latter to the sixth nucleus, termed the peduncle of the superior olive.

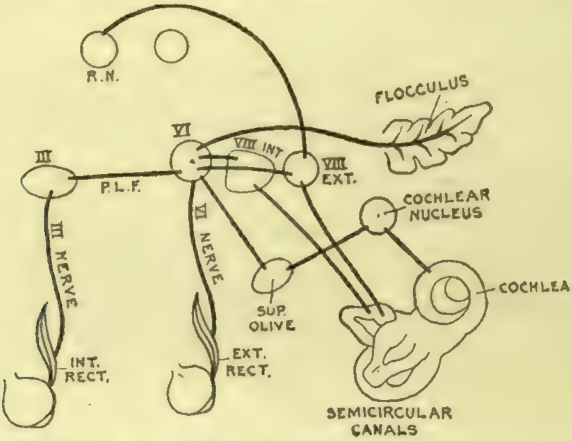

FIG. 11.—To show how the nucleus of the sixth nerve acts as the centre for conjugate deviation of both eyes to the same side, and also its connections with the auditory nuclei.

The Fifth Nerve.—The fifth nerve has a widely-extended origin in the medulla and pons. It arises by two roots, a motor and a sensory, the latter being much the larger. The motor root consists of the superior root of the fifth nerve, and the motor root proper. The superior (descending) root arises from peculiar vesicular, deeply pigmented cells which lie scattered at the lateral margin of the gray matter which surrounds the aqueduct of Sylvius. The fibres from these cells pass downwards in a series of bundles, which on transverse section form a crescentic group on the outer margin of the gray matter round the aqueduct. The bundles of fibres become gradually more anterior in their position as they pass downwards, and at the level of the motor nucleus they bend forwards and outwards to emerge with the motor root. The cells are seen among the fibres of the root throughout its extent, and become more numerous in its lower portion, forming a distinct group at the point where the fibres bend round to join the other roots. The main motor nucleus of the fifth nerve forms a group of large cells placed a little above the root of the seventh nerve, near the lateral angle of the fourth ventricle, on the inner aspect of the sensory nucleus of the fifth nerve. Its fibres arise from its outer aspect, and pass directly outwards and forwards to emerge from the lateral aspect of the pons at about the middle of its vertical extent. The *sensory* or inferior (ascending) root has a very extensive origin. It begins at the level of the second cervical root, and passes upwards throughout the cervical cord and the medulla, to the level of the motor nucleus of the fifth nerve, where it bends outwards, and emerges as a broad band of fibres external to the motor root. It is placed superficially, in the greater part of its extent, in the dorso-lateral part of the cord and the lateral part of the medulla. It is accompanied by an upward extension of the gelatinous substance of the posterior horn, which at the upper limit of the nerve becomes enlarged and forms the sensory nucleus of the fifth nerve in the pons. The fibres of the inferior root give off collaterals and end fibres which terminate in relation to this substantia gelatinosa.

The central connections of the fifth nucleus have not yet been clearly traced in man. In the rabbit an ascending tract passes from the cells of the sensory nucleus of the fifth nerve to the opposite side of the medulla. Here it passes upwards, at first anterior to the longitudinal fasciculus, but, at higher levels, within the lateral part of the tegmentum. This tract has been traced as far as the ventral nucleus of the optic thalamus.

The Seventh Nerve.—The nucleus of the facial nerve is situated in the anterior part of the tegment in the lower part of the pons, near its junction with the medulla. It lies between the superior olive and the inferior root of the fifth nerve. The root-fibres pass first backwards and inwards, forming a loop round the nucleus of the sixth nerve (knee of the facial), and then forwards between the facial nucleus and the inferior root of the fifth nerve, to emerge at the lower border of the pons immediately internal to the auditory nerve. Considerable difference of opinion still exists as to the origin of the fibres of the facial nerve which supply the muscles of the lips, and the orbicularis palpebrarum and frontalis. Gowers and others derive the fibres to the lips from the hypoglossal nucleus, on what appear to the writer to be insufficient grounds. Others, again, among whom the writer is one, derive the fibres for the orbicularis palpebrarum and frontalis muscles from a nucleus at a higher level, perhaps in the neighbourhood of the third nucleus. Both of these relationships, however, require further investigation.

The Eighth Nerve.—The eighth nerve has two roots, one derived from the cochlea, and termed the *anterior*, *superficial*, or *cochlear* root; the other derived from the semicircular canals, and termed the posterior, deep, or *vestibular* root.

The cochlear root terminates in the anterior accessory nucleus of Meynert (or cochlear nucleus) and the tuberculum acusticum, a narrow nucleus continuous with the former, and lying like a cap on the restiform body. From these nuclei secondary connections are formed through the corpus trapezoideum and the striæ acusticæ. The corpus trapezoideum sends fibres to the superior olives of both sides, to both posterior corpora quadrigemina, and to the opposite internal corpus geniculatum. This last-named structure is directly connected with the superior temporal gyrus by fibres which complete the central path for hearing. This is further strengthened by connections of each posterior corpus quadrigeminum with the internal geniculate body of its own side. The path of the striæ acusticæ is not so well established. They can be followed from the tuberculum acusticum inwards across the floor of the fourth ventricle to the raphé, where they pass directly forwards. According to Kölliker they pass round the pyramids of the upper medulla and enter the restiform body of the opposite side. This view still awaits confirmation.

The vestibular root enters the medulla between the restiform body and the inferior root of the fifth nerve. On passing the latter, its fibres, previously a compact strand, separate somewhat, and divide into ascending and descending branches. The latter form the inferior root, and can be traced as distinct bundles along the inner side of the restiform body as far as the cuneate nucleus, in which many of them appear to end. The nerve ends in the inner vestibular nucleus (dorsal or inner auditory nucleus) and in the outer vestibular nucleus. The latter has been subdivided into an upper (Bechterew's) nucleus, a middle (Deiters'), and an inferior nucleus. Of these the first is situated at the lateral angle of the fourth ventricle, the second opposite the point of separation of the entering nerve-fibres, and the third lies among the fibres of the inferior root itself. The inner vestibular nucleus lies internal to that first described, below the fourth ventricle, has a similar vertical extent, and presents on transverse section the appearance of a right-angled triangle with the apex at or near the raphé. The nucleus of Deiters is remarkable for the large size of its cells, which in this respect closely resemble those in the anterior cornua of the cord. Recent researches (Bruce, Held, Ferrier and Turner, Risien Russell, and Thomas) have shown that they form the starting-point for an important series of fibres which pass in various ways to the nuclei of the ocular nerves and to the cells in the anterior cornua of the cord. These fibres, so far as yet demonstrated, are as follow:— .

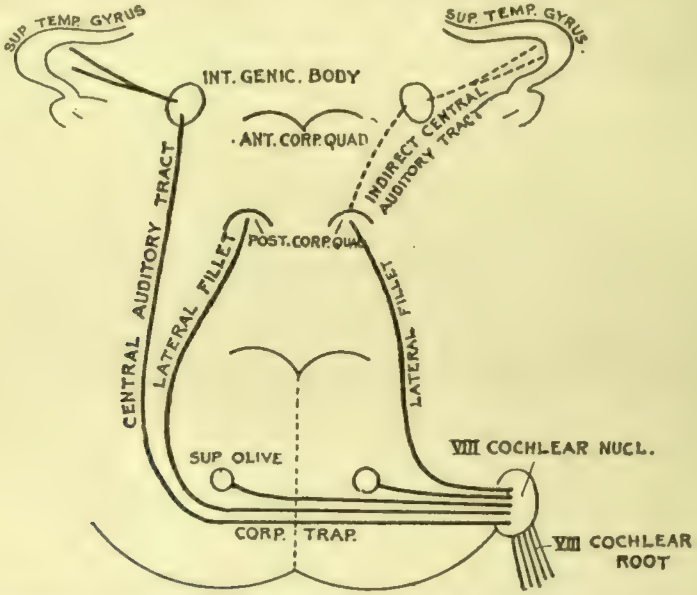

FIG. 12.—Diagram of the central paths for hearing. Shows the channels by which auditory impressions reach both hearing centres in the cerebral cortex.

1. A bundle which passes directly into the sixth nucleus of the same side.

2. One which, passing inwards under the fourth ventricle, enters the two posterior longitudinal fasciculi. In this some fibres bend upwards and

enter the two third nuclei, while others pass downwards within the cord and terminate in the anterior cornua of both sides of the cord (median vestibulo-spinal tract).

3. A large tract—the antero-lateral vestibulo-spinal tract—which passes obliquely inwards and downwards through the formatio reticularis round the outer side of the inferior olive, and then into the antero-lateral columns of the cord, from which at varying heights they enter the anterior cornua. They have been traced as far as the lower dorsal region by Russell.

4. A bundle which enters the opposite median fillet.

The nucleus of Deiters also receives from the opposite roof nucleus of the cerebellum an important strand—the cerebello-vestibular tract—at one time supposed to be a root of the auditory nerve, then termed the direct sensory cerebellar tract of Edinger, but recently shown by Ferrier and Turner to conduct from the cerebellum towards the nucleus of Deiters.

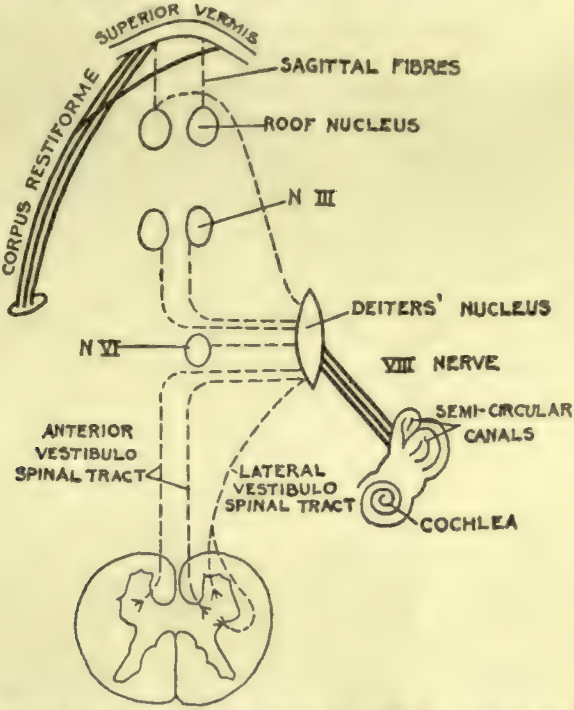

Fig. 13.—Diagram indicating the connections of the nucleus of Deiters. On the one hand with the semicircular canals and middle lobe of the cerebellum, and on the other with the third and sixth nuclei, and with the anterior cornua of the spinal cord.

The glossopharyngeal, vagus, and vagus-accessory nerves are arranged on the same plan, and arise along the whole length of the medulla. There are three roots, and three common nuclei:—

1. A motor root arising from the nucleus ambiguus in the middle of the formatio reticularis. It is probable that this root goes to the striped muscles supplied by these nerves.

2. A dorsal root (sometimes termed sensory, but almost certainly motor), arising from a nucleus of small cells in immediate proximity to the hypoglossal nucleus: behind the latter in the lower medulla, to its outer side in the upper medulla. As this root does not degenerate when the

nerve is destroyed by tumours, it is almost certainly efferent, and Marinesco has suggested that its function is to supply non-striped muscle.

3. The sensory root proper (fasciculus solitarius, or rotundus), which forms a rounded bundle external to the dorsal nucleus in the upper medulla, and behind it in the lower. This root terminates in cells which accompany it in its whole length.

The spinal accessory nerve is purely motor, and supplies the muscles of the palate, and larynx, and the trapezius, and sterno-mastoid. It arises in a special group of cells in the anterior cornu of the cord, which can be traced as low as the sixth cervical segment.

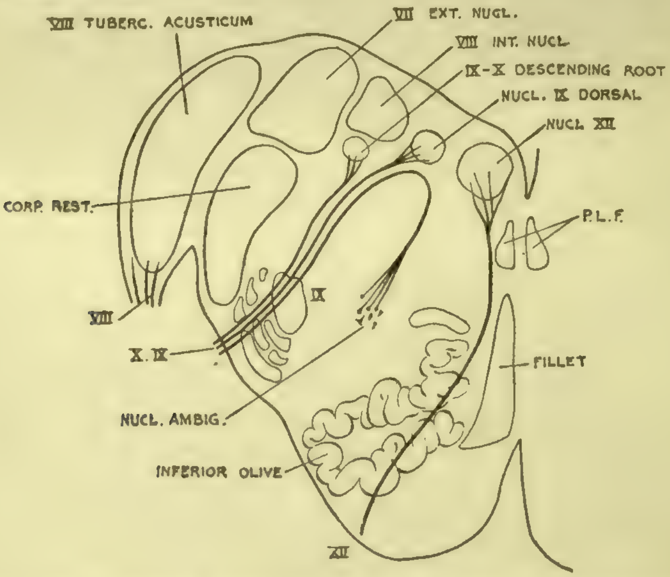

FIG. 14.—Diagram showing the origin of the hypoglossal, and vago-glossopharyngeal nerves.

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I. Anæmia of the Brain.—By anæmia of the brain is meant general cerebral anæmia, partial cerebral anæmia being almost invariably the result of some obstruction to the passage of blood through a particular vessel or group of vessels, on account of some local disease. General cerebral anæmia may be the result of anæmia affecting the whole body, anæmia dependent either upon a deficiency in the quantity of the blood, or, more generally, upon some change in its quality. The first of those conditions may be the result of severe loss of blood, either from injury or from visceral disease. The second is usually associated with very definite evidences of anæmia of other organs and other parts of the body. Anæmia of the brain also may be the result of some cause which hinders the access of blood to the brain through the usual channels. Thus, the pressure of an aneurysm, or of a growth on the carotid or vertebral arteries, may give rise to very marked anæmia of the brain. Cerebral anæmia also has been supposed to occur as a result of acute exhausting disease; but the evidence of this becomes each day less strong as we recognise more and more the likelihood that symptoms formerly attributed to anæmia in such conditions are more likely to be the result of the action of toxic substances on the brain.

Symptoms.—The symptoms of the condition vary with the degree of anæmia present, and also with the mode of its production. In the case of anæmia which is suddenly produced, drowsiness is a marked feature, the special senses are dulled, the pupils are at first contracted and may subsequently dilate, there is great muscular relaxation, the skin is pale and clammy, the respiration is sighing, there is a feeling of nausea, not commonly actual sickness, and consciousness may be lost. General convulsions, epileptiform in character, may occur. When general anæmia is slowly produced the brain action is usually excited with undue facility and with insufficient force. There may be dulness and drowsiness, although at times insomnia is a common result. In severe cases headache, sensory hyperæsthesia, *tinnitus aurium*, and vertigo may occur; and delirium is met with in extreme conditions. Convulsions are rare, and all the symptoms which have been mentioned are much more apt to occur when the patient is in the erect posture, and may be relieved by the recumbent position.

A set of symptoms has been described as the result of severe anæmia, the consequence, it may be, of diarrhœa or some other wasting condition occurring in children; the series of symptoms consisting in somnolence, with pallor, contracted pupils, occasionally rigidity of the neck, and depressed fontanelles.

When anæmia of the brain has lasted for a long time, it is certain, not only that the general nutrition of the brain suffers, but that the interference with the nutrition of cells and other structures results in actual anatomical changes—slight, it may be, but sufficiently severe to be recognisable. Thus the walls of minute vessels may be found degenerated, thickened, and homogeneous in appearance; the brain substance may be œdematous, and the nerve cells ill-defined and granular. Such conditions are associated with deficient mental power, and, if they occur in a child, may result in defective general development of the brain, leading to mental impairment, perhaps even permanent imbecility.

Pathology and Pathological Anatomy.—The general pallor of the brain, the changes in the cells, and other structures already alluded to, which are met with in severe and prolonged cases, sufficiently indicate the changes to be looked for in any condition suspected to be one of cerebral anæmia. But it must be remembered that pallor of the brain after death does not necessarily show that anæmia existed during life.

Pathology.—The exact conditions on which the symptoms described as those of cerebral anæmia depend are not known. Defective quantity and quality of the blood, with resulting interference with the cerebral structures, probably lie at the root of all the symptoms. It can easily be understood that if structures of such vital importance as those of the brain, the pons, and the medulla are either suddenly or slowly deprived of the material on which they depend for their nutrition, symptoms will arise depending upon interference with function. In a similar way, in conditions of general anæmia in which, as we have already said, not only are the structures imperfectly supplied with blood, but that blood which is supplied to them probably contains some toxic substances, it can easily be understood that symptoms depending not only on the defective nutrition of cells and fibres will arise, but also other symptoms more directly dependent upon the toxic effects of the foreign substances present in the blood. In this way, probably, are to be accounted for, not only the severe phenomena present in anæmia, such as convulsions, delirium, etc., but also those slighter phenomena, such as the tingling at the extremities, the headache, the nausea, vertigo, and interference with the special senses, which so frequently occur in such cases.

Diagnosis.—The diagnosis sometimes offers a certain amount of difficulty, but the difficulty consists not so much in recognising the anæmic condition of the brain giving rise to the symptoms, but in detecting the cause of the anæmia. If there has, for example, been any habitual loss of blood, such as results from menorrhagia or from bleeding piles, or even from frequently-repeated epistaxis, the recognition of the symptoms resulting from such loss of blood in its effects upon the brain need present no great difficulty. But certain anæmic conditions result not so much from such symptoms as from the effects of some deep-seated, obscure malignant condition present either in the bones or in some of the viscera; and the recognition that such conditions may be present is of the utmost importance. Nor must it be forgotten that other conditions strongly suggestive of cerebral anæmia may occur in people who appear anæmic, conditions which may not necessarily be the result of this anæmia. Thus it has happened that conditions of *petit mal* and vertigo, the result of quite other causes, have been supposed to be due to anæmia of the brain, although there was definite evidence that those symptoms were present long before the patient presented, at all events obvious, anæmia. But of course it is quite possible that cerebral anæmia may aggravate a condition of this kind which is already present.

Treatment.—The treatment necessarily resolves itself into a treatment of the cause of the anæmia. If dependent on a general systemic condition, such as pernicious anæmia, Hodgkin's disease, or similar disorders, the treatment appropriate to each condition must be carried out. One point of great importance in the general management is the recognition of the fact that rest in bed is often of the utmost importance. The symptoms resulting from cerebral anæmia are nearly always aggravated when the patient assumes the vertical position, and rest in bed in the recumbent posture is important, not only in order to allow the blood more easily to get to the brain, but also because it prevents, more effectually than any other line of treatment, demands upon an already weakened organism, which must result in aggravating the condition.

In regard to drugs, cardiac tonics, *e.g.*, digitalis, strophanthus, and arsenic, and hæmatinics, such as iron and arsenic, are among those most generally useful. Occasionally, in the extreme restlessness which is present in such conditions, it may be necessary to resort to opium; but it must be used with great caution because of the dangerous effect it may have upon the respiratory centre. In certain cases in which the symptoms are the result of sudden and profuse loss of blood, the transfusion of blood, or the injection of saline solution into the veins of the patient, may be urgently called for. In connection with the other remedies which have been mentioned, it is possible that the administration of small doses of nitroglycerine may be useful, because of the effect they may have in dilating the arterioles, and so permitting a more sudden and more complete access of blood to the brain.

HYPERÆMIA OF THE BRAIN.—In considering this subject great care must always be exercised by the observer not to *imagine* the pathological conditions to account for certain symptoms easily ascribed to hyperæmia of the brain. The very existence of hyperæmia of the brain, as related to any definite set of symptoms, has been doubted, and possibly with some reason. At all events, we may be quite sure that many of the conditions put down as the result of cerebral hyperæmia have no foundation in fact. Yet we cannot doubt that, as in other viscera, we may in the brain get a passive hyperæmia the result of obstruction, and it is also possible that we may have, as the result of anxiety, overwork, and of poisons circulating in the blood, a condition of the brain which may be justly enough termed active hyperæmia. And it is now recognised that some forms of recurring insanity have as their physical complement a condition of active hyperæmia of the brain.

Symptoms.—The symptoms ascribed to acute general hyperæmia are headache, tinnitus, sensations of throbbing, flushing of the scalp and face, and injection of the conjunctiva. It is doubtful, however, whether flushing of the face has any sort of correlation with flushing of the brain. Insomnia, even delirium with hallucinations, and other mental symptoms may be present; and dull headache, dizziness, and feelings of fulness in the head are common. It is quite impossible to particularise more than this the symptoms of such an indefinite condition. Probably any elaboration of details would only result in misleading or erroneous statement.

Etiology.—In reference to passive congestion, of course it is always produced by some mechanical obstruction to the flow of blood. Thus it occurs in heart disease, which causes overfilling of the venous system, and it may be produced by pressure upon any of the large veins in the chest or neck. Obstruction to the flow of blood through the lungs is also caused by chronic bronchitis and emphysema, and intensified by the act of coughing. It is said that slight congestion may be produced by the wearing of a tight collar. Active congestion may result from over-action of the heart; from sudden contraction of the arterioles elsewhere, such as occurs in the skin during a rigor; dilatation of arterioles, such as results from the use of certain drugs,—nitrite of amyl or alcohol, for example; and it may also constitute the first stage of an inflammation such as meningitis. Sunstroke is probably also a cause of active congestion, and symptoms ascribed to this have also been observed to occur in people who suffer from habitual loss of blood when for any reason this habitual discharge has been suppressed.

Pathology and Pathological Anatomy.—It is impossible to describe any definite pathological anatomy of such a condition, because of the fact that simple active congestion disappears after death in every organ. So that the lesson impressed by such a fact is that whenever such conditions as we speak of as congestion—namely, dilatation of small arteries, perhaps effusion of blood or serum round them—is met with in any case, such symptoms are more correctly to be ascribed to a commencing inflammation than merely to either active or passive congestion. Microscopical examination has shown the capillaries to be unduly large in passive congestion, and bulgings of them have occasionally been seen. After death by asphyxia minute vessels here and there may be found to have ruptured, and blood pigment has been found in the sheaths in cases of slighter long-continued congestion. Minute microscopic hæmorrhages into the cerebral substance may also be found almost constantly in those cases.

Diagnosis.—In the matter of diagnosis, it must be remembered that cephalic sensations alone are of no diagnostic value. There is little doubt that many cases of neurasthenia have been recorded as cases of cerebral congestion. It is really difficult to be definite in reference to diagnosis, but probably the truest guide is that which has been laid down by Gowers—namely, that the condition of congestion of the brain is rendered likely by the fact that the symptoms are increased when the amount of blood in the head is increased, and diminished under the opposite conditions.

Treatment.—In passive congestion, of course, the treatment resolves itself into dealing with the conditions giving rise to it. Many causes are, unfortunately, beyond our control; but in conditions such as cough, cardiac weakness, and others giving rise to the symptoms of cerebral congestion these symptoms may be, if not lessened, at least in some degree modified. When the congestion is an active one, attention to the conditions which allow of a free escape of blood from the over-filled capillaries, and to the assumption of postures, etc., lessening the amount of blood in the cerebral arteries, must be assiduously directed. If the symptoms are actually acute, and it is desirable to lessen the quantity of blood at once, resort must be had to venesection, to the use of leeches, or to rapid and violent purging. Blood also may be drawn to the limbs by immersing them in warm water, and it is often comforting, if not perhaps so useful as it is supposed to be, to keep cold constantly applied to the head.

Œdema of the Brain.—Œdema of the brain is defined by Mills as an effusion of serous fluid into or beneath the inter-cephalic membranes, or into the substance of the brain itself. It is doubtful whether its occurrence can be recognised definitely. It is extremely unlikely that œdema of the brain follows exactly the same laws as œdema of other organs, because we have in the brain very abundant lymph channels with ventricles and their connections, preventing probably in great measure actual effusion into the brain itself, except under conditions which are scarcely consistent with life. Of course, in speaking of œdema of the brain general œdema is meant, because partial or local œdema only arises in connection with local disease. It is likely that in severe conditions of hyperæmia of the brain some degree of œdema is always present.

Symptoms.—It is said that in conditions in which the effusion is general and infiltrating the intelligence is suddenly or slowly interfered with, according to the manner of occurrence of the œdema. Occasionally general convulsions are present, also twitching or rigidity, and even delirium. The œdema which accompanies senile wasting of the brain and which is compensatory does not give rise to symptoms, but it is likely that the only condition in which true œdema can be definitely proved is a condition associated with cerebral tumour, with intense intracranial pressure, and in such conditions the symptoms of the œdema are entirely masked by the general symptoms resulting from the presence of the intracranial growth. It is really useless to try to particularise the symptoms of cerebral œdema.

Etiology.—The etiology of the condition is sufficiently dealt with in what we have already said, and it may be stated generally that it is conceivable that œdema may occur in any condition in which there is hyperæmia.

Pathological Anatomy.—If the brain has been infiltrated with serum it is usually pale, softer, and wetter than usual, and on section of the substance a small quantity of serous fluid may be seen to run out. The fluid in the ventricles is increased, and the perivascular spaces are dilated and distended. These lesions, it will be seen, differ very little from the phenomena met with as a result of cadaveric maceration, and certain authors have regarded it as such.

The diagnosis and treatment have been sufficiently dealt with in what has been already said.

Cerebral Hæmorrhage

When cerebral hæmorrhage is spoken of, what is usually meant is bleeding occurring into the substance of the brain. Meningeal hæmorrhage, on the other hand, although it may sometimes also affect the brain substance, is usually spoken of under that name. Hæmorrhage may also occur into a cavity in the brain, one or other of the ventricles, and then it is sometimes spoken of as ventricular hæmorrhage. But from the clinical point of view all those three varieties—hæmorrhage on the surface of the brain, occurring originally from the membranes, hæmorrhage into the substance of the brain, and hæmorrhage into the ventricles, which may be primary or may be secondary to hæmorrhage into the brain substance—all three are so closely allied in their clinical symptoms that it is impossible to draw a hard and fast line between the different varieties.

Etiology.—Cerebral hæmorrhage is the result of the rupture of a vessel, usually an artery, and is almost always a result not only of conditions of pressure, etc., but also of disease of the vessel wall. Atheroma of arteries is a condition occurring as a result of increasing age and strain upon the vascular system. Such arteries undoubtedly rupture occasionally, partly on account of the unyielding character which atheroma imparts to the vessel wall, partly also on account of the conditions of high pressure, of which probably the atheroma itself is one of the effects. But it is most likely that cerebral hæmorrhage, in the sense in which we are at present considering it, is most frequently the result of the rupture of minute aneurysms, so-called miliary aneurysms, which form upon cerebral vessels as a result of weakening of the muscular wall, some even say of inflammation in it, and of increase in the blood pressure. It is very likely that atheroma of the large arteries may facilitate the production of such aneurysms by increasing the force and suddenness of the pulse-wave which reaches the smaller arteries. No doubt, also, the unsupported character of the cerebral vessels, in which such aneurysms are most apt to occur, facilitates the occurrence of such a change. Of the conditions associated with high blood pressure and the consequent occurrence of the changes alluded to in the blood-vessels, and of actual rupture of these, renal disease is by far the most important, and whether such renal disease is a cause of the vascular changes or a merely associated condition, its presence is of the utmost importance as a means not only of diagnosis, but also in reference to suggestive treatment.

It should also be mentioned that the endarteritis which is associated with syphilis may lead to rupture of vessels. This seems to be the case particularly in children who are the subjects of inherited syphilis, although no doubt the acquired disease has also similar results. Yet it must be recognised that, as a rule, the effect of endarteritis, as it occurs at all events in acquired syphilis, is to lead to thrombosis—to blocking of vessels rather than to their rupture.

As to the *exciting causes* of cerebral hæmorrhage, sudden and severe exertion of any kind, especially if it be unusual, is one of the commonest causes. Alcoholic over-indulgence has been immediately followed by the rupture of a cerebral vessel. It also has occurred during straining at stool and under excitement. But although all those factors have to be considered in reference to any particular case, it ought to be recognised that it possibly occasionally occurs during rest in bed. Cerebral hæmorrhage is rare under the age of forty, and it seems to become rather more rare during advancing years, so that between eighty and ninety it is far more uncommon, as we should naturally expect, than is thrombosis. It is rather more common in men than in women, but there does not seem to be any very marked disproportion.

SYMPTOMS.—In considering the symptoms of cerebral hæmorrhage, it must be understood that we are dealing here merely with those which occur at or in connection with the onset of the condition. In cases which recover from the initial stroke a permanent condition of paralysis is established. The various paralytic phenomena will be subsequently considered.

Manner of Onset.—The onset is sudden, and the patient is, as it were, struck down, as is indicated by one of the older and more common names—apoplexy. Consciousness may not be lost immediately, but is always impaired, and very often completely lost. Although at first it may be possible, to a certain extent, to rouse the patient so as to allow of swallowing being carried on, the condition of unconsciousness may deepen into profound coma. The time which it takes for this to occur varies in different cases, being determined—

(i.) By the amount of blood effused into the brain, and
(ii.) By the rapidity with which this effusion occurs.

Hæmorrhage into Brain Substance.—If, for example, the hæmorrhage is into the substance of the brain, on account of the conditions which are there present, it is apt to be slower, and consequently the symptoms are less sthenic in character. With such a condition the bleeding may be comparatively small in amount, and the constitutional effects, therefore, may not be very severe. It is even possible that consciousness may not be lost, although this is certainly uncommon. If it is not lost, then giddiness, with perhaps some headache, and the occurrence of paralysis, will mark the nature of the affection. It is rare for hæmorrhage in this position, unless associated with uræmia or some other toxic condition, to cause any convulsion.

Superficial Hæmorrhage.—Superficial hæmorrhage, on the other hand, which may be originally meningeal, is very apt to be associated with convulsion, starting in the limbs of the side opposite the side of the brain at which the hæmorrhage is taking place, spreading perhaps so as to affect also the other side of the body. Such hæmorrhage also, if convulsion be not present, may result in very considerable mental disturbance with delirium, perhaps associated with violence.

Hæmorrhage into the Ventricles.—Hæmorrhage into the other region to which we have referred, namely, into the ventricles, results in a much more serious initial condition than either of those to which we have already alluded. It can easily be understood that if hæmorrhage occurs into the ventricles, and if the blood is effused, as it is apt to be, quickly and in large quantity, that the condition quickly becomes one of the most extreme gravity. Such hæmorrhage usually results in immediate loss of consciousness, which rapidly becomes profound coma, and ends fatally in the course of a few hours. It may be primary, or it may be secondary to hæmorrhage occurring into the substance of the brain, the hæmorrhage extending, pushing apart the cerebral substance, and finally rupturing it, and so escaping into the ventricles.

Localising Phenomena.—Besides the general constitutional conditions there is, of course, a localised paralysis—a paralysis depending upon the position in which the hæmorrhage occurs. Hæmorrhage into the brain is almost always single, and its consequence is paralysis of the opposite side of the body, the limbs being affected in different degree according to the position of the hæmorrhage. If, for example, hæmorrhage occurs in its usual position—namely, in the anterior division of the capsule behind the knee—the paralysis is such as to cause more affection of the arm than of the leg, and to be unassociated with sensory disturbance. If, on the other hand, the hæmorrhage is farther back, then we are much more apt to have a condition present in which the leg suffers more than the arm, in which hemianæsthesia may be present and hemianopia is not rare.

PATHOLOGY.—The pathology of the condition has probably been sufficiently referred to in dealing with the ætiology. The pathological anatomy is concerned with the site, the appearances, and the subsequent changes in the effused blood.

(i.) *The Site.*—The two hemispheres are affected with nearly equal frequency, but of the various parts of the brain the central ganglia are undoubtedly the most common seat of hæmorrhage. It is significant that Charcot found that miliary aneurysms occur more frequently in this position than they do in any other part of the brain. Hæmorrhage into the centrum ovale may, of course, as already described, result in hæmorrhage into the ventricles. But it is certain that hæmorrhage primarily into the ventricles sometimes occurs, possibly from the choroid plexus, in which miliary aneurysms have been found. Hæmorrhage into the cortex is next in frequency, although much rarer than that variety to which we have just alluded. Hæmorrhage also occurs into the pons and into the cerebellum, but it is rare into the medulla and the crus cerebri.

(ii.) *The Appearances.*—When the blood is effused it forms a cavity, at first, by pushing aside the fibres and burrowing its way between them, and so pushing them apart. When this has reached a certain degree the fibres are ruptured, and unless this hæmorrhage is an extremely small one, it is rare for the fibres not to suffer with such severity as to be permanently damaged. Of course the blood may go on flowing into this cavity until some such accident occurs as rupture into the ventricle, and then we have a condition leading to sudden death. On the other hand, the hæmorrhage may be arrested at any particular stage, and then we have to deal with an encysted mass of blood, dark in colour and clotted.

(iii.) *Subsequent Changes in Effused Blood.*—The clot undergoes changes which eventuate in the formation of a cyst containing some straw-coloured fluid, fat globules, pigment, and other granules, as well as blood-crystals. The wall of the cyst, which was at first inflamed, also undergoes changes which result in the formation of connective tissue, of course at the expense of the nerve-fibres. Consequently we have a cyst formed which is in the nature of a cicatrix, in which a greater or smaller number of the nerve structures proper have been impaired or destroyed.

In cases in which the hæmorrhage has occurred on the surface of the brain the hæmorrhage may be widespread, but not large in quantity, and the resulting symptoms may be merely those of initial irritation, with perhaps some slight but temporary paralysis. On the other hand, the hæmorrhage may be such as to result in laceration of the brain substance, and consequent widespread and profound destruction of the cortical gray matter; but such conditions are much more frequently met with as a result of traumatism and consequent fracture of the skull than from spontaneous hæmorrhage. Hæmorrhage may also occur indistinguishable, so far as initial symptoms are concerned, from the ordinary form, into soft tumours, especially gliomata.

DIAGNOSIS.—*A. From Alcoholic Toxæmia.*—Probably the most serious mistake which may be made in reference to any case of cerebral hæmorrhage is in mistaking it for a case of alcoholic toxæmia. There is no doubt that the conditions closely simulate each other. Often the first aid given to a patient who has had symptoms such as are met with in cerebral hæmorrhage is to administer some form of alcoholic stimulant; and when the practitioner is confronted with a patient flushed, it may be dazed, or even unconscious, and strongly pervaded with the odour of alcohol, he may naturally enough be misled into regarding the case as one of simple drunkenness. But closer examination will usually reveal certain definite differences; and if the patient has, associated with those symptoms, conditions which favour hæmorrhage, or would suggest its occurrence, it is well to err on the side of safety, and treat the case as one of real illness. These conditions are, first of all, the presence of a high-tension pulse, with perhaps hypertrophy of the heart, and often detectable atheroma, even in a radial artery. If the urine be examined and be found to contain albumin, a still further confirmation is given to the likelihood that the condition is one of hæmorrhage into the brain. It is often difficult, especially if the patient is comatose, to definitely make out unilateral paralysis, but as a rule, even if the patient is comatose, a certain difference can be made out between the arms on the two sides in regard to their resistance to passive movement, for that on one side will probably be found to be much more flaccid than that on the other side. It will also be noticed that the stertorous, heavy breathing results in a flapping of one cheek as compared with the other, and that the pupils are usually small and often unequal. Besides those symptoms which enable one to recognise the condition that has to be dealt with, the patient's appearance may perhaps be of some help. It is usually the case that the flushing is marked, and the patient is perspiring freely; but this is not the invariable rule, for it does occasionally happen that the patient is excessively pale, and the skin cold and clammy. The condition of the pulse has been said to be usually a high-tension one, and this is undoubtedly the case in the initial condition. It may be that if the hæmorrhage has been large, and the condition is consequently one of collapse, the pulse may be small; but as a rule this is only a temporary phenomenon, and it very soon becomes high and rapid.

In regard to the breathing, it is often at first rather slow and sighing in character; as the coma deepens it becomes rapid and somewhat shallow, and it may in time—and this is usually of ominous significance—assume the Cheyne-Stokes' type.

B. From Thrombosis.—This is particularly important, not so much from its academic interest as from the different line of treatment that may be called for. The manner of onset has some diagnostic importance. In contrast with the sudden onset and absence of premonitory symptoms, so characteristic of embolism and hæmorrhage, we not infrequently find in thrombosis that headache, dizziness, or localised tingling sensations have been present for a short time previous to the attack. In thrombosis consciousness may not be lost, and the condition of the pulse is entirely different from that met with in hæmorrhage. The patient also, as a rule, will be older; yet it is by no means easy to differentiate those conditions, because the conditions of atheroma and arterial degeneration present in the one and leading to the condition of rupture of vessels, are also present in the other, and are conducive to the occurrence of clotting. So that the whole clinical picture has to be carefully considered with reference more particularly to the state of the heart and the other viscera, in order to determine which of the two conditions we have to deal with.

C. Uræmia or Diabetes.—Uræmia and diabetic coma may sometimes be mistaken for cerebral hæmorrhage, but in the latter we have the local symptoms, whereas in the former two conditions the symptoms are usually bilateral. Yet this is by no means invariably the case, and it will sometimes be found that a condition of uræmia results not only in unilateral uræmic convulsions, but also in a paralysis which affects one side more than the other, for which, nevertheless, no underlying anatomical change can be discovered.

D. Aneurysm, Tumours, etc.—The rupture of a large aneurysm, really of course a condition of cerebral hæmorrhage, may also closely simulate what we more strictly consider as cerebral hæmorrhage, but this probably has been preceded by symptoms indicative of intracranial growth, and further information which will assist in making a correct diagnosis of the condition will be found under this heading. We have already referred to the occurrence of hæmorrhage into tumours of the brain, but here also, although the onset may be exactly like that of an ordinary hæmorrhage, we will probably have a history pointing to the previous existence of the tumours. This, however, is not invariable, and it has happened to the writer to see a patient who was seized with right hemiplegia without any previous symptoms, in whom, after the lapse of some weeks, the evidences of tumour were conspicuous, and in whom the existence of a large gliomatous tumour was verified by post-mortem examination.

Prognosis.—The prognosis in any case of cerebral hæmorrhage is necessarily bad,—not, perhaps, in every case as regards life, but certainly in regard to complete recovery. If the hæmorrhage be a small one, of course the damage may be comparatively slight, and the constitutional effects not very severe; still, the fact that a hæmorrhage has occurred makes it impossible to foretell that the patient's life is safe for a single moment, because vascular conditions have to be recognised as present which are probably incompatible at all events with long life. On the other hand, if the hæmorrhage be large, more especially if there is any indication that it has burst into the ventricles, the outlook is of the most grave character, and it is almost certain that the patient will not live more than a few hours. In cases in which hæmorrhage has occurred on the surface of the brain, more especially if this hæmorrhage has been associated with traumatism, the prognosis is not necessarily bad if prompt measures be taken to deal with the affection. Hæmorrhage, however, into the basal structures, such as the pons and medulla, is necessarily attended with the gravest dangers on account of the proximity of vital structures.

Supposing, however, that we have to deal with a hæmorrhage which has become encysted, and which is undergoing the series of changes to which we have already alluded, there is no reason why we should not take a hopeful view in reference at all events to the immediate future. There is no doubt that such a hæmorrhage will cause some permanent damage, but one must recognise that the evidences of damage at first are much more severe than may afterwards turn out to be the case; so that whereas the leg and arm may be at first completely paralysed, we may find after the lapse of even a few days that a considerable degree of power has returned, on account, no doubt, of the fact that the affection of many of the fibres has been merely a temporary one, the result of pressure, with perhaps a slight degree of œdema. Yet it must be recognised that some of the fibres have been cut across, and that these will not be restored, that consequently there will be some permanent defect in the nature of paralysis of the limbs to a varying extent; and, looking further to the future, it must be recognised that there is always, in any case in which hæmorrhage has already taken place, a liability to the recurrence of a similar accident in the future.

Occlusion of Cerebral Vessels

The blocking of cerebral vessels is important from the character of the structures whose nutrition is interfered with. The effect, so far as regards paralysis, depends of course upon the area supplied by the affected vessel. Such occlusion of vessels may be divided into three classes: First, blocking of an artery by means of an embolus of a non-infective or infective nature; secondly, thrombosis or clotting occurring in an artery; and, thirdly, thrombosis in a vein or sinus.

I. *Cerebral Embolism.*—The most common source of an embolus is a diseased cardiac valve. Mitral disease, especially mitral stenosis, is by far the most common form of cardiac disease associated with embolism. Aortic valvular disease may also give rise to the same condition. An embolus also may originate from a clot which may have formed in the left auricle on account of some interference with compensation. This condition also is most like to arise in association with mitral stenosis, because of the incomplete emptying of the auricle which occurs in such a condition where compensation has been lost. No doubt, also, some condition of the blood rendering it more liable to clot favours the occurrence of such an accident. This is especially met with in the rheumatic state, and also during the pregnant and puerperal condition. In a similar way an aneurysm of any of the large vessels may be a source of an embolus which finds its way into the brain. A diseased or roughened vessel wall may also give rise to the formation of a clot upon it, and this clot, or a part of it, may become detached and lead to the obstruction of a smaller vessel more distant from the heart. Disease of certain kinds in the pulmonary vein may result in a clot passing from it into the circulation. In diseased conditions of the lung, also, septic particles may pass into the circulation through the pulmonary vein or its branches, and may reach the brain and there set up tuberculous disease, either meningitis or abscess. It is well known that an old-standing empyæma is one of the most common causes of abscess, and it is by some such mechanism as that just alluded to that such an abscess is formed. Although as a rule it may be that the other conditions enumerated as giving rise to embolism result in non-infective emboli, it will be understood that if the source of an embolus be a septic one, for example, if the endocarditis be of the ulcerative variety, the embolus from such a source may, and often does, set up changes at the spot at which it lodges in the brain which depend upon its septic character, and are quite independent of the usual mechanical effects. It has been stated that an inflammatory focus in the pelvis may give rise to embolism, yet it must be unlikely that any particle large enough to cause mechanical obstruction in a cerebral vessel could traverse the pulmonary capillaries uncaught, although it is true that these capillaries are larger than those, for example, in the brain. In certain cases, also, such a condition of cerebral embolism has arisen from some focus on the other side of the heart, on account of the accidental fact that the patient had a patent foramen ovale. Even without the interposition of such an anatomical peculiarity it is possible that minute organisms might traverse the pulmonary capillaries, but any subsequent cerebral complications would be more likely the result of infective inflammation rather than actual mechanical obstruction. Yet in most cases of such disease it is likely that the symptoms suggesting embolus are the result either of an embolism from a diseased cardiac valve, or of thrombosis occurring as a result of one or other of the conditions to be presently enumerated.

Local Results of Embolism.—Locally, embolism results, in the first place, in local anæmia. There is also œdema of the surrounding part, probably interfering with the function of a larger part of the brain than ultimately turns out to be diseased. There is also venous engorgement constituting the condition known as red softening. In some cases the collateral circulation seems to be such as to prevent any permanent impairment in the nutrition of the parts supplied by the blocked vessel, for recovery from a condition presumably of embolism in many cases is both rapid and complete. In the cases, however, in which such recovery does not take place, changes occur in the area supplied by the affected vessel resulting in the formation of a cyst with fluid contents containing blood crystals. Local changes also result in the vessel which is blocked. If the plug be of a septic or irritating character, inflammatory changes occur at the point where the embolus lodges, and these changes may spread into other vascular areas. If the vessel wall is weak or diseased, there may also result on the cardiac side of the plug the formation of an aneurysm, and it is not unlikely that in cases of embolic hemiplegia, in which death occurs suddenly some months after the first attack, this sudden fatal result is due to the rupture of such an aneurysm. The middle cerebral artery, or one of its branches on the left side of the brain, is the vessel most liable to suffer. The reason why embolism is more common on the left than on the right side of the brain probably is because an embolus may enter the left carotid more easily.

The symptoms of embolism are paralysis, which depends for its character upon the vessel affected. But the degree of paralysis, in most cases, is probably more severe at the commencement than it turns out to be subsequently, on account of the temporary impairment through œdema of a larger area of the brain than that primarily affected. Consciousness is usually, but not invariably lost; when it is not lost, it is probably because the vessel affected is a minute one. It is not often that the profound coma attending hæmorrhage is produced. Of course the chief indication in differential diagnosis between embolism and other conditions is to be found in the presence of some condition likely to give rise to embolism, especially cardiac valvular disease, although it must also be remembered that such a condition of cardiac disease does not necessarily exclude a diagnosis of hæmorrhage or of thrombosis. As to the clinical condition arising from embolism, that will be described when we have considered thrombosis.

II. *Arterial Occlusion or Thrombosis.*—The two conditions which have to be considered are diseased vessels and morbid blood states. Atheroma of vessels is by far the most common condition associated with thrombosis. It is, of course, a senile or degenerative change, and on account of the roughening which takes place in the wall this is apt to become the focus from which coagulation may spread, and so become the starting-place of complete occlusion. Of course this mechanism will only become effective under conditions in which the blood-stream is slow, or in which the blood has undergone certain changes which render it more liable to coagulate. The small cerebral vessels are peculiarly liable to suffer from atheroma, and the point at which the disease is particularly apt to start is that point at which a small vessel emerges from a larger one. This, of course, leads to a narrowing of the lumen of the smaller vessel, and it is at such a point that clotting is very likely to commence. Various conditions seem to favour an alteration in the blood, rendering it liable to coagulate. In old people it seems as if great or unusual exertion, or rather the state of exhaustion which is apt to follow such exertion, produced such a blood condition as renders it very liable to clot, for one of the commonest conditions in which thrombosis may occur is during the profound sleep which follows fatigue in those who are advanced in age. Chlorosis is another condition in which there is great liability to coagulation. But it seems likely that in such a condition the clotting is more apt to occur in the veins or sinuses. During pregnancy and the puerperium and also during lactation clotting may occur, and the blood states peculiar to gout and diabetes also favour this change. After typhoid fever we know that clotting in veins is apt to occur, and such coagulation may also take place in the cerebral vessels. After diphtheria hemiplegia occasionally occurs, and this is probably the result of cerebral thrombosis, although by some embolism has been invoked as the more likely cause. Yet there is little doubt that in the majority of such cases, at all events, no evidence of any endocardial change can be discovered, either at the time of onset or subsequently.

As to the local results of thrombosis, it is likely that œdema is not nearly so apt to occur, possibly on account of the condition of the blood and also on account of the disease of the vessel wall. There is, of course, in thrombosis not the same readiness for collateral circulation to be established, because the affected vessel is probably in the centre of an area, all the vessels of which are similarly diseased, so that the recovery from thrombosis is never so great as it may be from embolism occurring in young people. Besides œdema there is also venous turgescence, and consequently red softening, which may undergo changes resulting in yellow softening. But in some instances a widespread change takes place, especially if a large vessel have become affected, resulting in the formation of large yellow patches, the *plaques jaunes* of the French, scattered through the affected area.

Vascular Lesions of Syphilitic Origin.—Besides the thrombosis which occurs as the result of atheroma, we must also consider the condition arising as a result of the endarteritis of syphilis. This occurs in what is known as the tertiary stage, although one must be careful not to attach any time-limit to such a condition, for the writer has known it occur as early as six months after the primary infection, and certainly as late as ten years. So that by the tertiary stage we mean that stage of the disease at which new connective-tissue formations are apt to arise. The essential change in the vessels is proliferation of the intima, associated, as a rule, with some thickening of the adventitia. The essential process is considered elsewhere, but the effect of such overgrowth as concerns us in this place is a narrowing of the lumen, it may be a complete obliteration, of the vessel, and consequent interference with the nutrition of a certain area of the brain. The usual result is undoubtedly hemiplegia; yet if such a vessel as the basilar be affected we must have interference with the functions of all four limbs, of the facial muscles also, and perhaps of parts subserved by other cranial nerves on account of the interference with their nuclei to which such basilar disease leads. In many cases of so-called syphilitic hemiplegia the onset of the paralysis is sudden. Not uncommonly it is preceded for several days by severe headache, and the symptoms of general but indefinite illness, accompanied, it may be, with mental symptoms. Convulsion may also precede the onset of the paralysis, and this convulsion may be local. The paralysis at its onset may be accompanied by coma, which may last for several days, and, indeed, may result in death. Evidence of syphilitic disease elsewhere may be present, such as gummata in the liver, nodes on the bones, and such like. And there is very frequently to be detected marked thickening and hardening of the coats of the radial arteries, so that they feel like cords beneath the finger. The victims of such a condition are usually under forty, and in a condition of hemiplegia occurring in a man under forty without detectable cardiac or renal disease, one is justified in regarding the condition as most likely to be the result of syphilis. Unless such a case is met with in a very early stage and submitted to the most energetic treatment, the hemiplegia is likely to be persistent; and even with the most energetic treatment such a result cannot always be averted. It will be easily understood that if a vessel lumen has been gradually narrowed until only a very small patent passage remains, and when clotting has actually taken place in this little aperture, cutting off the blood-supply from the corresponding area of the brain, it is not likely that such a condition can be treated so as to lead to a re-establishment of the function of the vessel. Yet if such a vessel be small, it is quite possible, by energetic treatment in the early stage, so to affect other vessels which are similarly diseased, and whose occlusion will lead to much more severe symptoms, to avert the threatened danger. If, however, such a happy result is not obtained, the affected area undergoes necrotic changes similar in character to those attending other kinds of vascular occlusion. These we have already described. In addition to the cases of hemiplegia already described other clinical pictures are encountered. These are characterised mainly by the indefiniteness of the symptoms, the more prominent being persistent and intractable headache, usually associated with distinct mental alterations. Subsequent evidence of thrombosis is by no means uncommon in such cases.

In all forms of vascular occlusion occurring in the brain there is danger of death at the onset, and in many this actually occurs, the patient becoming comatose, Cheyne-Stokes' respiration coming on, and death supervening in the course of a few days or even a few hours. Of course this result is more likely to occur in cases in which a large vessel has been affected, and more particularly if the vessel affected be one like the basilar, subserving bilateral functions, and closely related to structures a healthy condition of which is essential to life. It may be stated as a general rule that occlusion of a vessel at the base is always more seriously significant than that of a vessel supplying any part of the hemisphere; so that in any paralytic condition it is always important to examine into the condition of the cranial nerves and muscles, because a definite affection of one or other of those structures may furnish important evidence as to the vessel which has become affected, and through that to the likelihood or unlikelihood of recovery taking place. And if a fatal result does not follow the onset of vascular occlusion at once, it will often be found that a period of danger arises at or about the third day, and again about the tenth day, when perhaps one is beginning to hope that all danger of a fatal issue has passed. What the reason of this is is not very clear, but of the clinical fact there is no doubt. (See also article "Syphilis.")

Paralysis Resulting from Vascular Lesions

The character of the paralysis resulting from vascular occlusion or from hæmorrhage will, of course, depend upon the function of the parts supplied by the vessels which have become blocked, or have ruptured, and of the parts which have been damaged in consequence.

Hemiplegia.—The ordinary form of paralysis is hemiplegia, that is, weakness of one side of the face and trunk and of one arm and leg. The whole of one side of the face is usually affected, although the weakness is most obvious in the lower part. The trunk affection is, as a rule, not well marked, although that also is occasionally distinct enough on the one side. The arm, in the great majority of cases, suffers more than the leg; and the explanation of those peculiarities is to be found in the hypothesis known as Broadbent's. According to this, bilaterally associated movements are represented on both sides of the brain, and the closer the bilateral association the more nearly equal is the representation on the two sides of the brain. Thus it is well known that the two lower limbs are much more closely associated in their movements than the two upper. One arm is frequently used quite independently of the other, while a movement of one leg without some participation in that movement by the other leg is comparatively uncommon, and of course in the habitual use of the lower limbs for purposes of locomotion the association is a very close one. The same is true to even a greater degree of the movements of the trunk, for it is impossible for most people to move one side of the chest or abdomen without moving the other. Facial movements, also, are nearly always bilateral. The two sides of the forehead especially are very closely associated, the orbicularis palpebrarum less so, although some people find it impossible to close one eye without closing the other. In regard to the parts about the mouth, the association, although close, is not quite so close as that between the two sides of the upper part of the face. Yet it is closer than between the two legs, and still more close than that between the two arms. Now, if we imagine a vessel to be occluded in such a position as to affect the face, arm, leg, and trunk areas or fibres equally, the arm on the opposite side will be most affected, the leg less affected, the face still less so, and the trunk scarcely at all, at all events in an obvious or permanent way.

Pseudo-Bulbar Paralysis.—Similar considerations explain the phenomena of the condition known as pseudo-bulbar paralysis or double hemiplegia. In this condition swallowing and articulation are affected. The movements subserving those actions are strong in their bilateral association, and consequently, according to Broadbent's hypothesis, in their bilateral representation in the brain; so that a lesion affecting one side of it, if it affects them in any noticeable degree, only does so slightly and temporarily. If, however, in addition to the lesion causing unilateral weakness we have superadded a lesion producing weakness of the opposite side, it will at once be seen that considerable interference with these actions is not only likely but almost inevitable, so that with such a double lesion we have a condition similar in many respects to that produced by true bulbar paralysis.

Paralysis with Sensory Disturbances.—Resuming now consideration of the usual form of paralysis resulting from vascular rupture or vascular occlusion, viz. paralysis of one side of the face, body, and trunk, such a paralysis may be purely motor. If, however, the area subserved by the posterior cerebral artery be affected, anæsthesia of the paralysed side of the body, with affection of the special senses also on this side, may result, because of the affection of what is known as the sensory crossway lying in the posterior part of the internal capsule, the part supplied by the posterior cerebral artery. In such a condition hemianopsia may also be present, that is, blindness of the opposite halves of the field of vision, opposite, that is, to the lesion in the brain. And when we have hemianæsthesia and hemianopsia in any case of hemiplegia, the leg is likely to suffer more than the arm, because the leg fibres of the internal capsule are posterior to the arm fibres, and therefore presumably nearer to the diseased area. A similar condition as regards vision may also arise from the affection of a vessel supplying the occipital lobe. (See page 9.)

Aphasia.—When the lesion causing hemiplegia is on the left side of the brain, speech is apt to be interfered with so as to result in some form of aphasia (*q.v.*) In left-handed patients a right-sided brain lesion may also give rise to this, while a left-sided lesion in such a patient may not result in aphasia. Dr. Hughlings Jackson has also pointed out that in cases of right hemiplegia, when the patient is not left-handed, and when there is no aphasia, the leg, as a rule, suffers more than the arm; while the converse is also true, that in a patient with right hemiplegia, in whom the leg suffers more than the arm, aphasia is not so likely to result. The reason for this, of course, lies in the considerable area intervening between the leg area or fibres and the speech area or fibres, as compared with the small space which separates the hand or arm area or fibres from those subserving speech functions.

Rigidity.—Besides the actual powerlessness or paralysis which is present in the limbs on the affected side in hemiplegia, there is also usually rigidity: first, the initial rigidity occurring at the onset of the paralysis, ascribed to irritation of fibres; second, secondary rigidity coming on after the lapse of weeks, or it may be months; and, third, permanent contracture in which actual structural changes have taken place in the muscles, and perhaps in the joints. This is really the late resulting condition of secondary rigidity. It is not always equally marked, for often in the early morning the limbs are less stiff; and one curious phenomenon is, that often when the patient yawns the hand or arm, at other times rigidly contracted, may open or relax and become momentarily limp. The explanation of the rigidity is still obscure. Dr. Hughlings Jackson ascribes it to unantagonised cerebellar influx, and this seems by far the most feasible explanation, although it is not easy to understand why it should not be established, on such a hypothesis, from the very commencement when the cerebral influence is cut off.

The Reflexes.—Along with this rigidity there is increased activity in the deep reflexes. The knee-jerk is unusually active, not only on the side paralysed, but also on the other. It is really more correct to speak of both sides being weakened, one considerably more than the other. And so it is also with the knee-jerks; both are increased, that on the weaker side more than that on the other. Often, also, there is a rectus clonus, and a foot clonus may be present on the paralysed side at least. The wrist and elbow reflexes are also increased, and often also the jaw-jerk. The superficial reflexes are said to be usually diminished, but there is change to be noticed in most cases in the plantar reflex at least, namely, that the contraction of the toes which follows stimulation of the sole is not, as it usually is in health, a flexor spasm, but an extensor spasm. Usually at first there is some difficulty with the bladder, namely, in passing water, and retention may result unless it is relieved. This, however, is usually a transient phenomenon. Constipation is common, but rarely is there incontinence of fæces. Swallowing may be difficult at first, and articulation may be interfered with. But except in cases in which there is a lesion on each side of the brain, such difficulties are usually only temporary.

Vascular Lesions of Crus and Pons.—The foregoing are the conditions met with usually in cases in which the cortex or the white matter, or the internal capsule, are diseased. When the lesion affects the crus cerebri, there is in addition to the hemiplegia an affection of the third nerve on the side opposite from the hemiplegia, that is, on the same side as the lesion. This may be complete or incomplete. If the region affected is the upper part of the pons, the hemiplegia is of the usual type, namely, face, arm, and leg on the side opposite the lesion. If, however, the affected area is the lower part of the pons below the point where the tract subserving facial movements crosses to its nucleus, the paralysis is what is known as crossed or alternate hemiplegia, the face being affected on the same side as the lesion, the limbs on the opposite side. In such a condition the external rectus muscle on the same side as the lesion is also liable to be paralysed. Similarly, there may be wasting and weakness of the tongue on one side, and of the lips on the opposite side from the lesion in the vicinity of the hypoglossal nucleus of one side; but such a case of crossed hemiplegia is exceedingly rare.

Thrombosis in Cerebral Veins and Sinuses

The thrombosis to which we have directed attention in the previous pages characteristically occurs in arteries. The veins and sinuses of the brain are, however, also occasionally the seat of a condition of thrombosis, which gives rise to important, sometimes grave symptoms. Such thrombosis is either simple non-infective, the result of some general condition; or, secondly, infective, the result of some local contamination with septic material.

1. Simple Thrombosis.—This is most common in children in the first six months of life, generally as the result of acute diarrhœa or some other wasting disease. On this account it has been called "marasmic." It usually affects the longitudinal sinus, although it may extend into one or two of the neighbouring sinuses as well. The cerebral veins also may suffer, and under certain conditions, for example after an attack of typhoid fever, a vein may be the only vessel blocked.

A determining condition of such thrombosis is no doubt the weakening of the blood-stream, the loss of fluid from the blood, and, in consequence of those two conditions, liability for clotting to occur. After acute illness, such as pneumonia, pleurisy, etc., when the more severe symptoms have subsided, such a condition may also occur, and it may be generally stated that the exhaustion and prostration of severe, long-continued, and debilitating illness are the conditions which favour the occurrence of the thrombosis which we are considering.

Morbid Anatomy.—Sometimes the affection is only of one sinus, the longitudinal. Sometimes, however, others are involved, and veins also may be affected. In fatal cases this has usually occurred. Occasionally only a surface vein seems to be affected, giving rise to focal symptoms, namely, unilateral convulsions and weakness. The clots are dense; they do not, as a rule, completely fill the lumen; they tend to become organised and absorbed, and rarely disintegrate. There is evidence of congestion in the cerebral veins and capillaries, and extravasations occur, minute but numerous, from the latter. Œdema also may result. The ventricles may be distended and the fluid may spread into the retro-ocular tissues and give rise to exophthalmos. The condition of the brain resulting from this is not definitely known; it is probably one of induration and atrophy.

Symptoms.—These are not usually definite enough to permit of a diagnosis being made. An indefinite illness of cerebral type, especially if it have been ushered in by a convulsion, and if it have followed prolonged and exhausting illness, should suggest sinus thrombosis. If œdema of the scalp be present, associated with localised convulsions, such a condition will suggest an extension of the thrombosis so as to implicate a cortical vein.

The treatment best adapted to the relief of the condition is by the copious imbibition of stimulating fluid. If, however, diarrhœa is still present such treatment is inadmissible. It will then be necessary to resort to a large injection of tepid water into the rectum, or to the injection of saline fluid into the veins. As soon as the patient is able to tolerate fluids by the stomach these should be administered copiously, and as hot as the patient can take them.

Another variety of simple or non-infective thrombosis of veins or sinuses is that which occurs in chlorotic girls about the age of twenty. The longitudinal sinus in this class also is most frequently affected. The clots are usually firm, but they may be softened, and there are usually also present hæmorrhages from engorged veins or capillaries. The onset of the symptoms is preceded, as a rule, by a period of unusually hard work. Severe headache for several days is the immediate precursor, and this is frequently accompanied with vomiting. Convulsions also may come on, either unilateral or bilateral, succeeded by paralysis of one side. When this takes place the condition usually ends in coma and death. Recovery occasionally takes place from such a condition, but then the patient remains hemiplegic. Optic neuritis is common in the condition, and it is possible that many of those obscure and unexplained cases of optic neuritis occurring in anæmic girls, associated with severe headache and vomiting, are the results of thrombosis occurring in some of the veins or sinuses closely related to the optic nerve.

2. Infective Thrombosis.—This variety is secondary to a focus usually in the middle ear, occasionally in the nose or in the tonsils, or in the face or orbit. An anthrax pustule has been known to be the primary lesion. Compound fractures of the skull, also, in which the wound has become septic, may be the starting-point. When the sinus is affected the wall becomes denuded of its epithelium, and the consequent roughness determines the point at which the thrombosis will start. The wall then becomes thickened and infiltrated, so that hæmorrhage from this is not to be feared as a rule. But by this infiltration the area of the process may

become indefinitely enlarged. Collections of pus may form, or general meningitis may result, and the existence of such a clot constitutes a danger of distant infection, more especially of the lungs, where septic emboli may set up abscess, pneumonia, or even gangrene. Sometimes, however, the clot may have no tendency to break down, although the process may extend, and in such cases we may have a vein like the jugular completely blocked and to be felt as a hard cord along the anterior border of the sterno-mastoid. The general features of septic sinus thrombosis are fairly well marked, although the symptoms are not very definite.

The Diagnosis is facilitated by the recognition of an infective focus. The local symptoms naturally vary with the sinus affected. The symptoms are characterised by severe headache, rigors, profuse sweating, anorexia, diarrhœa, and an irregular temperature. Œdema of the scalp over the thrombosed sinus is frequently present. Lung symptoms are often absent, except some cough and occasional sharp pain during the first twenty-four hours after the lung infection. But then the prune-juice expectoration and the presence of râles soon place the matter beyond dispute. The offensive odour of the secretion and also, it may be mentioned, of the patient's stools, render extensive deodorising necessary. Pain in the liver may also be present, probably from extension of the inflammation to the diaphragm. The lungs become extensively disintegrated, perhaps studded with abscesses. The mental condition may remain unimpaired up to the very end. Such is *the pulmonary type of* infective thrombosis. An *abdominal* or typhoid type has also been described, with pain, diarrhœa of a pea-soup character, very offensive, with meteorism, thus closely simulating typhoid fever. These symptoms usually come on about the second week, and are accompanied by a dry, foul tongue, a measly kind of rash—quite distinct from typhoid rash—pallor, languor, headache, muttering delirium, and great prostration.

In a third class of cases the symptoms are *cerebral*, or rather meningeal, consisting chiefly in excitement, agitation, twitching of muscles, squints, head retraction, and delirium. This is rather an unusual type of sinus thrombosis, and it may be complicated by the occurrence of cerebral abscess. The treatment of this affection is considered on page 97.

Conditions Resulting from Vascular Lesions

There are some conditions which are occasionally present in the course of hemiplegia, either at the onset of the illness or in later stages, which must be referred to.

Athetosis.—Amongst these one of the most interesting is that form of mobile spasm known as athetosis. In this condition the paralysed limb or limbs, although the arm is the part chiefly affected, undergo a cycle of slow movement, one position gradually passing into another, none of the movements being jerky. Although the arm is the part chiefly affected, this spasm is not by any means restricted to that, for the leg and foot may also be affected, and also the face. This athetosis occurs far more frequently in cases of hemiplegia of which the onset has taken place in early life, than in those in which the paralysis has occurred later. But it is met with occasionally after the attacks which occur in later life, and probably more frequently in those which are traumatic in their origin (*vide* "Athetosis").

Other Forms of Involuntary Movement.—Besides this athetosis or mobile spasm, another form of involuntary movement also occurs after hemiplegia. This has been spoken of as post-hemiplegic chorea, an unfortunate name, which has not even the advantage of being descriptive. In this condition the movement is a jerky, sharp movement, usually increased by attempts at voluntary motion, and therefore resembling in some ways the so-called "intentional tremor" of disseminated sclerosis. This form also is more frequent after lesions occurring in early life, although not absolutely restricted to them.

Convulsions.—Convulsions in association with hemiplegia occur at two stages: first, in connection with the onset of the paralysis, when the convulsions are presumably to be ascribed to the initial irritation depending upon the lesion; and, secondly, after the paralysis is completely established, when they are to be ascribed to some persistent irritation around the focus of damage. In the former case the phenomena may be merely transient. In the latter case, when they have once occurred they are apt to return, and have to be treated on the same principles as ordinary epileptic attacks. In both cases the convulsions commence on one side, and are often restricted to the paralysed side, although not invariably.

Contractures.—Contractures are very apt to occur in the paralysed limbs. These, of course, depend upon the preponderating activity of certain groups of muscles, and it is exceedingly difficult altogether to prevent their occurrence. Probably the most troublesome and most important contractures occur in the hand and wrist. This subject is further referred to in the article "Deformities."

Trophic Changes in the Skin, Muscles, and Joints.—These are of two kinds, according as they occur in the initial stage of the disease or as a later phenomenon. Of the former, undoubtedly the most important and the most ominously significant is the acute sloughing bed-sores, which may occur in the first few hours of the onset of the paralysis, and is very apt to escape the notice of the nurse unless she has been warned of the possibility of its occurrence. This bed-sore occurs over the paralysed side, affecting the tissues over the trochanter and over the sacrum, and may penetrate in the course of a few hours so deeply as actually to lay bare the bone. The bed-sores which occur at a later stage of the illness are not so significant and not so troublesome. It is sometimes difficult to entirely prevent their occurrence, but much can be done by the care and cleanliness which are bestowed upon the patient (*vide* "Bed-Sores").

Besides bed-sores, trophic changes in the skin and the other parts, often associated with changes in the joints, which themselves may be the result of interference with nutrition, are apt to occur. Joint changes occur most frequently in the shoulder, and occur more frequently on the left side than they do on the right, as was long ago pointed out by Hughlings Jackson. This joint change may result in almost complete immobility of the shoulder joint, and is followed by diminution in the size of the muscles and often in actual pain along the nerve trunks, and in some cases there is evidence that the joint change has spread so as to involve the cords of the brachial plexus, and we have then in such cases the usual evidences of brachial neuritis, namely, wasting of muscles, tenderness of nerve trunks, glossy skin, and alterations in the conditions of sweat secretion.

TREATMENT

The treatment in any given case of paralysis must be divided into two parts, according as we consider the treatment at the onset of the attack and the treatment necessary when the paralysed condition has become established.

TREATMENT AT THE ONSET.—*In Cases of Hæmorrhage.*—It is evident that the condition of a patient who suffers from even a small rupture of a small vessel, especially if it occurs, as it usually does, associated with a high-tension pulse and with diseased vessels, may be one of extreme precariousness. It is difficult to say that the hæmorrhage will not rapidly enlarge, and give rise to a condition which immediately threatens the patient's life. Anything, therefore, in the way of treatment must be both prompt and energetic, and of course it is evident that the first thing to do is to put the patient under conditions in which the least possible voluntary effort will be necessary. Besides putting the patient in bed, it is often desirable to put him, not in the recumbent, but in a semi-recumbent posture, in order as far as possible by position to minimise the effect of the blood-pressure. If the pulse be high-tension and bounding in character, it is evident that something must be done to lower this tension. In the old days, no doubt, such a condition would have been treated by energetic bleeding, and it is almost a pity that this treatment has fallen so much into disuse, because by it we can probably effect more quickly than by any other means the sudden and profound lowering of arterial pressure which is really desirable. Should, however, bleeding not be possible, one must have recourse to more gentle methods with a similar object, namely, purging, especially by means of saline cathartics. Thus, if two or three grains of calomel are administered at once, if a castor oil and turpentine enema be given, and if, after the lapse of some hours the bowels have not acted, another enema be also given, we shall probably have such an effect upon the vascular system as will result in very considerable lowering of blood-pressure, and the consequent establishment of conditions which tend to stop any hæmorrhage that is taking place. It is also desirable to get the skin and kidneys to act freely by measures which will at once suggest themselves, and it is essential to use every other means to reduce the blood-pressure as has already been pointed out. The application of cold to the head may not be very effective, but if the patient have even a slight degree of consciousness it is often comforting.

Suppose that the patient has survived the initial stage, and has regained consciousness, we then probably have to deal with very severe head pains; and there is little doubt that such head pains are very much relieved by some form of counter-irritation, and the best, in the writer's experience, is by means of leeches applied over the temple or behind the ear. If leeches be, for any reason, inadmissible, then blistering over similar regions may possibly be followed by a similar effect.

With regard to food, care must be taken that the patient receives a certain amount of nourishment in the most bland and easily digested form possible, and yet as little fluid as he can do with. Milk and milk foods are undoubtedly the best, both from their readily digestible nature and from their being less liable to contain ingredients that might, by their toxic character, raise the blood-pressure. A milk diet should always be maintained for at least several days after the acute stage is over. Such measures are, of course, intended to counteract the conditions which have given rise to hæmorrhage; and when one is satisfied that the hæmorrhage has ceased the condition is still somewhat precarious, and must remain so for several days, because a vessel which has given way so recently is not likely to be very firmly sealed. So that the patient must be kept quiet, his diet must be bland and nutritious, and the bowels, the kidneys, and the skin must be kept acting freely.

In Cases of Embolism.—As regards the condition occurring when embolism has taken place, what has been said as regards general principles in reference to hæmorrhage must of course hold true in reference to embolism. The same kind of diet is necessary, rest is just as essential, because the dangers that have to be avoided are any further strain upon the heart—already probably weak—and the setting free of another embolus. Of course the cardiac condition must be treated, if treatment be necessary. If there be dilatation, for example, this will be treated *secundum artem*. If weakness and irregularity, the ordinary heart tonics should be employed.

In Cases of Thrombosis.—In thrombosis we have an entirely different condition to deal with. Here the symptoms depend upon clotting occurring in an artery, the result either of a weak blood-stream occurring in diseased arteries, or of a peculiar condition of the blood rendering it prone to clot, or of a combination of those two. It is evident that rest in such a condition is also necessary, in order especially to prevent too great demands upon a heart already perhaps overstrained. In reference to other points, however, an entirely different mode of treatment must be adopted from that at all events pursued in a case of hemorrhage. The patient must be stimulated, best probably by means of fluids. Often strychnia is necessary, and that can most efficaciously and with the best effect be administered hypodermically. It is well to avoid anything like too free purgation, because of the effect in favouring coagulation which might ensue. Yet it is desirable, undoubtedly, that the bowels should be actively opened in order that the blood may be changed in its character, and so, perhaps, the tendency to clot got rid of. What has been said about food holds good in thrombosis also. It must be bland, nutritious, and not too abundant, and, as we have already hinted, in this condition it probably ought to be chiefly fluid.

If the thrombosis or stoppage have occurred in a vessel which is the seat of damage by syphilitic disease, the treatment of course must be most energetic. And although the general principles, to which we have alluded, of treatment in thrombosis must be followed, there must be associated with this the active administration of iodide of potassium, and along with this of mercury. The latter in such cases especially is undoubtedly most efficaciously administered by means of inunction, and the patient must be as rapidly as possible brought under the influence of the drug. This will be indicated by the condition of the gums, and when he is once under the effect of mercury this ought to be kept up for some time in a mild degree. As we have already said, it does not follow that because a vessel is blocked by syphilitic disease this can be entirely got rid of by the most energetic treatment, and disappointment is often met with in what are apparently promising cases. Still, in every case in which there is a likelihood of the disease being syphilitic, the patient must have such energetic treatment as we have described, in order, if possible, to get rid of the diseased condition of the vessel walls which renders thrombosis so easy.

Such, then, are the general principles on which treatment should be carried out at the onset of an attack of hæmorrhage, embolism, or thrombosis.

Treatment of Paralysis.—We must now direct attention to the measures which are necessary for the paralysis which is found to be present when the initial stage has been passed.

We may begin by stating that, as a general rule, in all cases of hemiplegia there is a tendency for recovery to take place to a certain extent. In the great majority the leg improves more than the arm, and in very few cases of hemiplegia is it the case that walking power remains entirely absent. So that, however apparently hopeless any particular case is as to locomotion in the early stages, one is always justified in holding out hopes to the patient of his being able to get about. The most troublesome thing is the contracture which is apt to take place in the arm. This can usually be obviated, at all events to a great extent, by early commencing to use massage and passive movements. Sometimes, also, it is useful to electrify the extensor muscles of the forearm, because flexor contracture is what is most apt to take place, and stimulation of the extensors will to some extent tend to prevent the over-action of the flexors (*vide* "Deformities"). These really embody the general principles on which any case of hemiplegia should be treated. As regards bed-sores, and other trophic changes which occur during this stage, these, of course, are a matter of nursing and dressing according to ordinary surgical principles. There is no doubt that in cases of intractable bed-sores the internal administration of opium favours the healing of those. In reference to the other trophic changes, that occurring about the shoulder is very difficult to deal with, because the pain in the early stage is often so great as to prevent passive movement being carried out. If adhesion have taken place it may be necessary to administer an anæsthetic, such as nitrous oxide gas, and forcibly move the arm at this joint. If, further, there is evidence of involvement of the brachial plexus, anodyne applications, such as glycerine and belladonna, or even the hypodermic injection of morphia may be necessary, for the condition is one of great pain, and is apt to very much reduce the patient's strength. When the pain has subsided the use of galvanism with massage, in order to stimulate the impaired structures, is necessary.

As to treatment by drugs, of course much is not to be expected from the use of those in favouring recovery. But ordinary tonics, such as strychnia, digitalis where thrombosis has been the initial lesion, and these combined with others when the cardiac condition in cases of embolism has been such as to demand them, embrace the drugs which are most commonly necessary. Of course if convulsions occur they are, as a rule, fairly easily controlled by means of bromide. But one must be careful that the dose used is not too large, otherwise grave mental phenomena, transient usually, may supervene.

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ETIOLOGY.—Like tumours in other situations, there is much that is obscure with regard to the causation of those of the brain, but we are nevertheless in possession of certain definite facts bearing on this question. Some cerebral tumours are congenital in origin. The rare instances of *dermoid cysts* and *teratomata* belong to this group, while *syphilomata* are sometimes congenital, and probably even gliomata may arise in this way in exceptional cases. From what has just been said, it follows that heredity may be operative in the causation of syphilomata, and there is reason to suppose that the same factor may play a part in the etiology of tubercle and carcinoma.

No period of life is exempt, except it be the first six months, which supply no clinical evidence of the existence of tumour. The largest number of cases are met with between puberty and the middle period of life, while only a limited number occur in people over sixty years of age. *Tuberculous tumours* are most common in children, although the growth may be of this nature even in an aged individual. *Gliomata* and *sarcomata* are most common between puberty and middle age, though both forms are met with at other periods. *Syphilitic* tumours usually occur during the fourth and fifth decades, while carcinoma belongs to the middle and later periods of life. *Parasitic tumours* are most common in the second and third decades, and are very rare at any other periods.

Males are more frequently affected than females, a state of things that has been attributed to certain causative factors of cerebral tumour, such as syphilis, injuries to the head, alcoholic excess, and mental strain being more commonly operative in men than in women. This explanation cannot be regarded as wholly satisfactory, however, in that preponderance in the male sex is met with in children as well as in adults, so that while some or all of the above factors may aid in swelling the number of males as opposed to females affected in adult life, an explanation has yet to be found which will satisfactorily account for a preponderance of males being attacked among children as well as among adults.

Trauma has a distinct influence in the generation of a new growth. Though probably in some cases ascribed to this cause, the growth had been in existence prior to the accident, which only served to evoke more rapidly manifestations which must, nevertheless, inevitably have appeared without the aid of the injury. On the other hand, years may elapse after the trauma before any manifestations of the tumour are forthcoming. It is obviously difficult to estimate to what extent is justified the belief that alcoholic excess, mental strain, depressing emotions, or a general state of ill-health are to be counted among the causes of brain tumours.

The station in life appears to play no part in causation, except in so far as the poorer classes are more exposed to the influences of syphilis, tubercle, and trauma, and are more liable to attack by cysticerci and echinococci.

Where the tumour is syphilitic or tuberculous there is commonly evidence of similar affection in other parts of the body. Metastatic growths are rare, carcinoma being more usually of this character than sarcoma, and the lung is the most common seat of the primary growth. In the rare cases in which parasitic cysts occur in the brain, others of a like nature may be met with in other parts of the body.

MORBID ANATOMY.—Any form of *new growth* may be met with within the cranial cavity, though the frequency with which different forms occur naturally varies. Tuberculous, syphilitic, gliomatous, and sarcomatous growths are the most common, carcinomata are less frequent, fibromata and osteomata still less so, while cysts, both simple and parasitic, endotheliomata, psammomata, cholesteatomata, lipomata, and neuromata are the rarest forms of tumour. A *hypertrophic enlargement of the pituitary body*, though not a new growth, nevertheless constitutes a tumour, and is liable to give rise to the manifestations common to intracranial tumours; so too *large intracranial aneurysms* must be similarly classed.

Tumours of very varied sizes are met with either singly or as multiple growths. They originate in the bony walls of the cranium, the meninges, or some part of the brain, crura cerebri, cerebellum, pons, and medulla oblongata, and may be found on the surface or deeply buried in one or other of these parts of the central nervous system. They may be well defined and easily demarcated from the surrounding brain tissue in some cases, while in others the morbid passes insensibly into the normal, so that the microscope can alone determine where morbid ends and healthy begins.

The bone may be rarefied in consequence of the increase of intracranial pressure; but the reverse of this is commonly met with, the bone being unusually dense and hard. The *meninges* are thickened, and it may be adherent where a neoplasm reaches the surface of the brain, and in some instances limited thickening of the meninges is seen at some part remotely situated from the tumour; but widespread meningitis does not occur except in cases of syphilitic and tuberculous growths.

A certain amount of *softening or œdema* of the parts surrounding a tumour is commonly seen, and the growth may itself be softened in consequence of degenerative changes, which may ultimately lead to disintegration and cystic formation. An accidental condition which may be met with is softening of some area of the brain remote from the seat of the tumour, consequent on obliteration of a blood-vessel through pressure by the neoplasm. *Hæmorrhages* are frequently seen in and about the growth, and in some cases so extensive is the extravasation that the microscope can alone definitely determine the dual nature of the morbid condition present.

In its growth a tumour may insinuate itself between the nerve elements, doing wonderfully little active damage to them in the process. The more solid forms of growth compress the nerve tissues, and then a process of slow atrophy or more rapid disintegration results in accordance with the rate of growth of the tumour.

Dilatation of the ventricles is a common feature, especially when the tumour is subtentorial in situation, and flattening of the convolutions of the cerebral hemispheres is an accompaniment, the sulci being obliterated to a greater or less degree and the convolutions widened.

Pathology.—Towards a due comprehension of the problems which have to be considered in relation to the pathology of cerebral tumours, it is essential to remember that in the adult, at any rate, the skull is practically a closed cavity whose walls are rigid and allow of no expansion. It thus follows that anything added over and above the normal contents of this cavity, which is already full, must of necessity make room for itself at the expense of all other structures contained in the cavity. Tumours accordingly occasion deleterious effects by their *pressure* on the brain tissue, the nerve roots, and the meninges. They no doubt make some room for themselves by destroying the nerve elements in the part they are growing, but sooner or later the tumour grows out of proportion to the amount of local destruction which it causes, and thus parts at a distance must of necessity suffer from its pressure. A tumour in one cerebral hemisphere does not merely exert its influence on that half of the brain, but, in virtue of the imperfect septum provided by the falx, it makes its effects felt on the opposite hemisphere also. So too a tumour situated below the tentorium of necessity produces the most serious consequences on adjacent structures, such as the cerebellum, pons, medulla, and certain of the cranial nerves, but it also has a serious effect on the cerebrum, in spite of the septum provided by the tentorium, owing to the increase of fluid in the lateral ventricles which is especially liable to occur in these cases. This leads me to speak of another way in which the contents of the skull are hampered for room in some cases of cerebral tumour, viz. by *increase of the fluid in the lateral ventricles*, consequent on dropsical effusion, the result of impediment to the return flow of blood from the choroid plexuses through the veins of Galen, together with interference with the exit from the ventricles. The lateral ventricles dilate to accommodate the increasing amount of fluid, their increase in size, of course, taking place at the expense of the brain tissue. These pressure effects, however, induced, of necessity derange the functions of the nerve elements, and are responsible for many of the symptoms to which cerebral tumours give rise. That to the increase of intracranial pressure is to be largely ascribed the general symptoms which result in these cases is proved by the fact that on trephining the skull, and thus relieving intracranial pressure, these general symptoms either disappear or are much mitigated. Leonard Hill's experimental observations lead him to conclude that the brain does not transmit pressure equally in all directions, and that the cerebro-spinal fluid cannot transmit a local rise of pressure to other parts of the brain. This observer regards any pathological increase of cerebral pressure as of circulatory origin; there is, according to him, a rise of capillary to arterial pressure at the seat of lesion consequent on obliteration of veins and capillaries by the foreign body. Moreover, it is not to mechanical pressure, but to cessation of blood-flow that he attributes the symptoms of compression, and anæmia of the spinal bulb is regarded as the cause of the major symptoms of this kind.

The question of the relation of increase of intracranial pressure to optic neuritis is very complicated. Some attribute the condition of the optic papilla to increase of intracranial pressure, while others contend that the neuritis is the result of the spread of a process of tissue irritation to the optic nerves, which sets up an intense inflammation when it reaches the papilla. Neither view alone is wholly satisfactory; for, on the one hand, attempts to increase intracranial pressure experimentally fail to produce optic neuritis, while, on the other hand, relief of intracranial pressure by trephining is followed by subsidence of the neuritis when there is no reason to suppose that any irritative process acting before is not still in operation.

There are symptoms which may persist in spite of any measures adopted with a view to relieve intracranial pressure; they are of two kinds, the one pointing to some local disturbance of function, while the other is indicative of a widespread derangement. The former group of symptoms is to be ascribed to the *local effects* of the neoplasm, whether those effects be of an irritative or destructive nature; in short, they depend on direct interference with the nerve elements by means of the tumour. The latter group, on the other hand, appears to depend on some *irritative condition* which spreads far beyond the limits of the neoplasm, either owing to disturbance of the vascular and lymphatic circulations in the brain, or by the dissemination of irritative products derived from the new growth

SYMPTOMS.—The symptoms to which intracranial tumours give rise may be conveniently considered in two groups, general and localising, though no hard and fast line separates the two, for under certain circumstances one or other of the general symptoms may have a localising value. The manifestations are usually gradual in onset; but it sometimes happens that the onset is sudden, and in some cases, but for the possible detection of optic neuritis, the phenomena are indistinguishable from those which result from vascular occlusion or rupture. General symptoms are such as are common to intracranial tumours, irrespective of their precise seat, while localising symptoms are those which indicate more or less definitely the actual seat of the neoplasm.

Of the *general symptoms*, headache, vomiting, and optic neuritis are prominent, while convulsions, giddiness, and mental disturbance belong to the same category. It must not be supposed that all of these symptoms, or even the first three mentioned, of necessity occur in every case of intracranial tumour, for on the contrary tumours may exist without engendering symptoms of any kind, or while giving rise to some evidence of their presence, one or more of the cardinal symptoms may be absent.

Headache.—Of the general symptoms none is more constant than headache; indeed it is only in exceptional cases that this symptom is absent. The pain varies in character and intensity, but is sometimes most agonising. Such severe pain occurs in paroxysms, and in the intervals there may be complete freedom from pain; more commonly, however, some pain, moderate or slight in degree, persists even in the intervals between the severe paroxysms. It is usually deep-seated, though sometimes superficial, and is commonly increased by muscular efforts, coughing, or other conditions which cause *passive cerebral congestion.* In some cases the pain is troublesome when the patient wakes in the morning, in which case giddiness and vomiting may accompany it. The pain may be general, or it may be localised to one part, or even when general it may be more intense in one part than in any other. Localised pain may correspond to the seat of tumour, but too much reliance cannot be placed on this evidence, as while it is of undoubted value in some cases, it leads to serious errors in others. *Tumours on the surface* of the brain are especially liable to give rise to localised pain which corresponds in situation with the seat of the tumour. In addition to this, *tumours of the frontal lobes* commonly occasion frontal headache, while *tumours of the cerebellum or occipital lobes* frequently give rise to occipital headache; in other words, the seat of the tumour and that of most pain correspond. There is, however, no constancy with regard to this; for, in addition to cases in which the pain is equally intense in all parts of the head, there are those in which, with a tumour situated anteriorly, the pain is chiefly complained of in the occipital region, and others in which, with a tumour situated posteriorly, the pain has been referred to the forehead. Cerebellar tumours commonly give rise to occipital pain, but on the other hand the pain may be frontal, in which case it may be either on the same side as the tumour or on the opposite side. The crossed frontal headache in cerebellar tumour has been regarded as of localising value by Purves Stewart, who considers that it is to be accounted for by the anatomical connection between one lateral lobe of the cerebellum and the opposite frontal lobe of the cerebrum.

From the point of view of localisation, more important than pain is *localised tenderness* on pressure or percussion of the skull.

Vomiting, though common, is not so frequent as either headache or optic neuritis, nor is it usually so early a symptom as headache. In some cases it is most intense and frequent, in others it perhaps only occurs once or twice throughout the whole course of the case, while in some again it may be a prominent symptom during a certain period of the case, without being present either before or after. There may be the most intense feeling of sickness, resulting in strenuous efforts at vomiting, and such attacks may be induced by the slightest movement of the head, while, on the other hand, the most copious vomiting may occur without provocation, and with very little nausea. Like headache, the vomiting is paroxysmal, and indeed the two are often combined, such attacks, it may be, lasting days, or even weeks, to be followed by periods of complete cessation. In other instances a severe attack of headache culminates in vomiting, it may be with complete relief of the pain. The vomiting is not related to the ingestion of food, though it, of course, frequently happens that the attack comes on at that time. The appetite remains natural, and the tongue is usually clean, though in some cases it is furred, more especially when vomiting has been going on for some time. Although it may be a symptom of tumour in any situation, vomiting is most frequent and severe in *subtentorial tumours;* indeed this symptom is rarely absent in these cases.

Optic Neuritis.—This sign occurs in a large proportion of cases of intracranial tumour. Though not so common as headache, it is not often absent through the whole course of the malady, and is of much greater value in diagnosis than headache or any of the other general symptoms, all of which may be met with in hysterical affections, whereas optic neuritis is positive proof of organic disease. So important is the detection of optic neuritis that an opthalmoscopic examination should habitually be made, at least in every case in which severe headache is a symptom. Both eyes are usually affected, unilateral neuritis being a rare condition, but inequality in the degree of neuritis on the two sides is not so infrequent; moreover, the neuritis may begin and remain limited to one eye for some time before the other is also affected. Marked optic neuritis may exist without diminution in the acuity of vision or limitation of the visual fields; but with the development of consecutive atrophy there is progressive loss of sight. The acute process may reach its maximum in a few weeks, while in other cases it takes many months to do so. Tumours in all situations are not equally prone to produce optic neuritis; those which press upon some part of the convexity of the cerebral hemispheres, especially where the growth spreads itself out more or less flat, are less liable to do so than those which infiltrate the brain. So, too, *absence of optic neuritis* is relatively frequent in tumours of the crura cerebri, corpora quadrigemina, pons, and especially the medulla oblongata. As a rule optic neuritis is only of value in the general diagnosis of cerebral tumour, but intense double optic neuritis of rapid onset with marked swelling, numerous hæmorrhages, surrounding retinal changes, and functional disturbance of vision is suggestive of a tumour of the cerebellum or one of the optic tracts, especially in the neighbourhood of the optic chiasma. The second of these positions is made certain if hemianopsia or unilateral blindness is present. It is not a little singular that tumours in this same situation may cause blindness and optic atrophy without any neuritis. *Optic neuritis which is unilateral*, which begins on one side, or which is considerably more intense on one side than on the other, is also of some localising value, as the tumour is usually in the cerebral hemisphere on the same side, and while there are cases in which the tumour is in the opposite hemisphere, this is less likely to be the case when it is in the anterior part of the brain, so that with evidence that the tumour is situated anteriorly, the probabilities are that it is on the same side as the excess of neuritis. The intensity of the optic neuritis is no certain criterion of the size of the tumour, for though the larger the tumour the greater the pressure and the optic neuritis, inexplicable exceptions occur in which very large tumours produce no optic neuritis, and very small ones are accompanied by marked neuritis. The neuritis may develop rapidly in a chronic tumour, but as a rule such a rate of progress is indicative of rapid growth of tumour, progressive hydrocephalus, or meningitic complication.

Vertigo is much less common than any of the symptoms already considered. Various abnormal sensations are, however, described as giddiness by patients which do not strictly deserve to be classed as vertigo. Neoplasms occasion this symptom through *interference with the auditory nerve* in some cases, while in others they do so through the agency of *ocular palsies*, induced by interference with the nerves supplying the ocular muscles, or the nuclei from which these nerves originate. In the absence of evidence of either of these mechanisms severe vertigo is strongly suggestive of a tumour of the cerebellum, its middle peduncle, the corpora quadrigemina, pons, or medulla.

Convulsions.—Two forms of convulsions may be the outcome of a tumour of the brain, *general convulsions* in no way distinguishable from those which characterise idiopathic epilepsy, and the *localised convulsions* known as Jacksonian epilepsy. The former variety is common to intracranial tumours in general, irrespective of their precise seat, and therefore has no localising value. Such convulsions may exist for years, and the case be regarded as one of idiopathic epilepsy until the detection of optic neuritis reveals the real nature of the case. General convulsions appear to depend on increase of intracranial pressure, or on cerebral irritation engendered by the presence of the tumour; but in some instances they are to be accounted for by hæmorrhage into or around a tumour. However induced, a general epileptic seizure, or a series of these, may lead to a fatal termination. A further resemblance to idiopathic epilepsy may be seen in attacks of sudden loss of consciousness without convulsions, which simulate petit mal.

In a *Jacksonian fit* we have a symptom of considerable localising value, for as a rule such convulsions are indicative of a tumour in or contiguous to some part of the sensori-motor cortex of the Rolandic area of the brain.

Moreover, such convulsions, though they may ultimately become general, and thus indistinguishable from the variety already described, have a local beginning, and may remain localised to some part throughout the attack. On the other hand, when the convulsions spread from the part in which they commence, they do so in *definite order*, and, as a rule, with more or less deliberation. They are due to irritation of the centres in the cortex which preside over the movements of the parts that are in a state of spasm. This may further mean that the tumour is situated in or near that part of the cortex, a significance that they have as a rule, though by no means invariably. The exceptions are supplied by certain cases such as those in which the convulsions commence in the thumb and fingers, although the tumour is situated in some part of the cortex removed from the hand area. The explanation of this discrepancy appears to be that the cortical centres concerned with such highly specialised functions as those subserved by the thumb and fingers are more readily disturbed than those related to less specialised functions. Thus it is that although fits which begin in the thumb and fingers may indicate a tumour in the part of the cortex concerned with the movements of those parts, those which commence in the lower extremity, or in one of the proximal segments of the upper limb have a greater and more absolute localising value. Moreover, when the damage to the cortex is sufficient to cause *paralysis*, it is the surrounding less damaged part that is prone to discharge, so that in such cases the paralysis is a surer guide to the seat of tumour than the initial convulsion. Apart from such persisting paralysis, *temporary paresis* or *paralysis* often follows a Jacksonian fit, and is most marked in the parts first or most convulsed. A further important feature of this variety of fit is that consciousness is not usually lost until the convulsions become bilateral.

Tonic Spasm or Contracture.—Apart from the rigidity which results in the limbs in cases in which these are paralysed, tonic spasm is most commonly met with in *subtentorial tumours*, and in some of these cases the spasm in the neck muscles is sufficient to cause some retraction of the head. The muscles lower down the spine may share in the spasm, and paroxysms of general tetanic rigidity may occur.

Athetosis.—Except as an accompaniment of hemiplegia (an unusual one in tumours), athetosis is rare, and has usually been met with in neoplasms in the region of the optic thalamus.

Tremor and inco-ordination have also been symptoms of tumours in the same situation, but are more characteristic of growths in the cerebellum, though those of the corpora quadrigemina and pons may also be attended by like phenomena. Pressure on the pyramidal path by a tumour of the pons or crus has been known to give rise to a coarse, jerky tremor, like that seen in disseminated sclerosis, and, as in that affection, has only been evoked on attempts at voluntary movement.

Apoplectic attacks may result from hæmorrhage in connection with a vascular tumour, while in other cases the attacks are rather pseudo-apoplectic, a sudden accession of intracranial pressure from some cause, vascular disturbance which occasions œdema or congestion, or irritative inhibition by the tumour possibly accounting for some of these seizures. However induced, these attacks may result in death.

Mental disturbance of some sort or other is common in cases of intracranial tumour, especially in the later stages of their course. A variable amount of *hebetude* is the most common condition; little interest is taken in his surroundings by the patient, and questions have to be repeated more than once before a reply can be elicited from him. All degrees of this mental apathy are met with up to actual coma, but on rousing such a patient sufficiently his replies are of a character which prove that there is no real perversion of his intellect. In some cases there is *alteration of manner*, in others *irritability*, or *loss of memory*, or *emotional disturbance*. True insanity is rare, but melancholia, mania, and dementia all occur. It is not improbable that the true explanation of such cases is that the tumour serves to evoke the manifestations of the particular variety of insanity in one predisposed. Cerebral tumours that give rise to mental symptoms may be unattended by the more usual phenomena by which intracranial growths are recognised, so that such patients are sent to asylums, where the real nature of their malady is unsuspected until the end is approaching, or is only revealed on necropsy. It is probable, however, that fewer of these cases will go so long unrecognised when the ophthalmoscope comes to be more habitually used in all asylums. Hysterical manifestations may certainly be evoked in this way, and may lead to error in that the organic affection may be overlooked in the presence of undoubted evidences of hysteria. In some cases the clinical picture resembles that of a person suffering from *general paralysis of the insane*.

Much difference of opinion exists as to whether tumours of the frontal lobe are more liable to give rise to psychical disturbance than those in other situations. My own experience leads me to believe that *mental deterioration is especially common in frontal tumours*, but that to be of any value in localisation two reservations are essential. The mental defect must be manifest early in the history of the illness, and something more than mere lethargy must be present. Hebetude is, however, not without some value if it is a symptom early in the history of a case, at a time when it seems reasonable to conjecture that there is not as yet much increase of intracranial pressure. There is no reason to suppose that true insanity is more liable to occur with tumours in this situation than with those in any other part of the brain, though a mental state comparable to that met with in general paralysis of the insane is sometimes observed in frontal tumours. The mental disturbance is oftentimes very slight, a mere oddness of manner, a peculiar childishness, irritability, or some other form of change of character, failure of mental power leading to mistakes in manual as well as in more purely mental operations, failure of memory, and the like.

LOCALISING SYMPTOMS.—*Aphasia*.—It is remarkable how seldom either motor or sensory aphasia which is pronounced, and which persists, is produced by a cerebral tumour. Reduction of a patient's capabilities of speech to "yes" and "no" probably never occurs as a result of an uncomplicated tumour, though such complete aphasia may, of course, result where the neoplasm is complicated by hæmorrhage or softening. Tumours tend to displace the nerve elements to a greater degree than to destroy them, in addition to which such destruction as results takes place gradually, and there is thus time for substitution and compensation to come about through the agency of parts unaffected in the same and in the opposite cerebral hemisphere. On the other hand, marked but fleeting aphasia not uncommonly follows a convulsive attack, and some aphasia, either motor or sensory, may form part of the clinical picture if the tumour is situated in or near any of the cortical areas of the left cerebral hemisphere which are concerned with speech processes.

Paralyses of various kinds are met with, and most of them are of considerable localising value. *Hemiplegia* may of course result from tumours in very different situations, provided they be so placed as to be capable of exerting a deleterious influence on any part of the motor path from the cerebral cortex to the spinal cord; but graduated paralysis, as a rule, points to the Rolandic region of the cortex as the seat of mischief, and, moreover, this variety of paralysis is a more certain indication of the seat of tumour than is localised convulsion. Hemiplegia and monoplegia are the common varieties of paralysis that occur; bilateral paralysis involving either the two lower extremities (paraplegia) or all four extremities in which the arms suffer more than the legs is rare. The latter variety of paralysis results when the tumour is situated in a part where the motor paths are in such close proximity as to allow of their being both interfered with by a single lesion, so that the pons and medulla supply such cases. It is also said that tumours of the cerebellum by pressing on the pyramids may cause this combination of paralysis; but I question the truth of this, excepting when a tumour of the cerebellum invades the pons or medulla secondarily. When a hemiplegia is present it may be of the ordinary type, or less commonly one or other of the crossed varieties may be met with, the latter depending on a lesion of the crus cerebri, pons, or it may be cerebellum. The paralysis resulting from the presence of a cerebral tumour may be progressive and permanent; on the other hand, as has already been said, that which follows an epileptiform attack is fleeting, but it sometimes happens that without such attack paresis occurs temporarily, probably as a result of some œdema or other vascular disturbance in the immediate vicinity of the tumour. Localised paralysis may, of course, result from pressure on, or involvement of, one or other of the motor cranial nerves.

Cranial Nerves.—Few of the localising symptoms supply more trustworthy evidence than affection of certain of the cranial nerves. Unfortunately, however, there is scarcely one of them that is not liable to succumb to some general influence, possibly increase of intracranial pressure, so that faulty evidence of the seat of a tumour may be thus supplied. We are prepared for deleterious effects from general pressure in the case of a small nerve with a long intracranial course, but in that large nerves may also suffer, there is a possibility that a neuritis comparable to that which affects the optic nerves may prove to be the cause at least in some cases. Much as the affection of individual nerves is thus robbed of value as an aid in localisation, the affection of some of them in certain combinations is almost free from this drawback, especially combinations in which parts of the functions of some of the nerves are alone affected, in which case a central defect may be regarded as almost certain. *Paralysis of the third nerve* results from tumours in the region of the crus. The *fourth nerve* is usually affected in combination with others, the growth as a rule involving the corpora quadrigemina. Least reliance can be placed on paralysis of the *sixth nerve* as a localising sign, as one or both nerves may be affected in tumours of very different situations; but they are specially apt to suffer when the growth is subtentorial. If *all the nerves supplying the muscles of one eye* are paralysed, the tumour must be in the region of the *sella turcica*, and sight is usually lost in the same eye. *Paralysis of the external rectus* of one eye, and of the *internal rectus of the other*, results from tumour of the pons, in which case the eyes cannot be turned towards the side of the lesion, and the facial nerve may be affected on the same side. The *fifth nerve* is involved by tumours in or outside of the pons, and in addition to paralysis in the distribution of its motor and sensory branches there may be loss of taste. Like the fifth, the *facial* is also paralysed by tumours in or outside of the pons, in the former case in conjunction with the sixth, and in the latter with the eighth. The *spinal accessory* is paralysed by tumours in the region of the medulla oblongata, and if the neck muscles are affected the growth must be outside the medulla. *Paralysis of the hypoglossal* results from tumours in very much the same situation as those which affect the spinal accessory; indeed the two nerves commonly suffer together.

Sphincters.—It is exceptional for paralysis of the sphincters to occur as a result of a cerebral tumour. Most of the accidents which happen in which the patients pass their evacuations under them are to be explained by the amount of mental hebetude present; but, on the other hand, exceptional cases are met with in which there is loss of control over the sphincters which cannot be thus explained. *Tumours of the pons and medulla* disturb these functions in a more direct way than do tumours in other situations, though loss of control over the sphincters has been sufficiently frequently met with in frontal tumours to make the fact noteworthy.

Sensory Disturbance.—Some form of subjective sensation, in the sphere of ordinary sensibility or in that of one of the special senses, may be the initial phenomenon of an epileptiform attack, in which case it is the sensory disturbance, and not the subsequent convulsion, which is of significance as regards localisation. *A general blunting* of sensibility which is met with where there is much mental hebetude is to be attributed to this rather than to any involvement by the tumour of parts of the brain concerned with sensory processes. *Hemianæsthesia* occurs when the posterior part of the internal capsule is pressed on or invaded by a growth, in which case hemianopsia may be an accompaniment. Tumours of the gyrus fornicatus also cause a similar disturbance of common sensibility. Apart from such gross defects, slighter loss is especially liable to occur when the sensorimotor cortex of the Rolandic region is involved. There is very slight blunting of sensibility to tactile impressions most marked in the parts in which there is most motor defect, in addition to which impressions that are perceived are erroneously localised. When the fifth nerve is involved, anæsthesia in the distribution of one or all of its branches may be forthcoming, prior to which, however, there may be hyperæsthesia, or spontaneous neuralgic pains of similar distribution.

Sight.—Phenomena indicative of irritation of the *optic nerves,* such as flashes of light and the like, are exceedingly rare, except when they result from irritation of the half-vision centre in the occipital lobe, when they constitute the auræ of convulsive seizures, and are usually referred to the visual field on the opposite side to that of the tumour. From what has been said when optic neuritis was under discussion it is clear that loss of sight is an important and common symptom of intracranial tumour. This defect may, however, result, without optic neuritis, from direct implication of some part of the optic path by the tumour. Instead of a general defect of vision there may be only loss in some parts of the fields constituting one or other of the varieties of hemianopsia, the most common of which is *bilateral homonymous hemianopsia,* which indicates either involvement of one optic tract, the fibres of the optic radiation in their passage to the half-vision centre in the occipital lobe, or the centre itself. It occasionally happens that a tumour presses on the *middle of the optic chiasma,* in which case bilateral temporal hemianopsia results. Thus while blindness the result of optic neuritis is of no localising value, it is otherwise when this loss is unattended with neuritis.

Smell.—Anosmia may be a symptom of an intracranial tumour in any situation, but is comparatively rarely met with. Considering the proximity of the olfactory bulbs to the frontal lobes, it is singular how seldom loss of smell is a symptom of tumours of this part of the brain; nevertheless anosmia does occur in some cases, and if unilateral, is in favour of the tumour being on the side on which the loss of smell is detected. Anosmia also occurs in connection with *cerebellar tumours,* though not as frequently as was at one time supposed. The mechanism by which it is induced when a tumour is situated so far back is obscure, unless it be due to neuritis of the olfactory nerves comparable to that which affects the optic nerves.

Taste is rarely affected, though, as has been said, loss of taste may be a symptom when the *fifth nerve* is destroyed.

Hearing.—Subjective sensations indicative of irritation may arise from interference with the auditory nerve by *tumours of the pons* or *base,* in which case the sensations are referred to the ear on the same side, or they may be induced by growths in the *temporo-sphenoidal lobe,* in which case they are referred to the ear of the opposite side, and may constitute the aura of an epileptiform attack. Deafness results from damage to the auditory nerve by tumours at the base of the brain, and also by those which destroy the nucleus of the nerve in the pons. Bilateral deafness is said to have been produced by *tumours of the corpora quadrigemina involving the tegmentum.* A variable degree of deafness, usually slight, sometimes considerable, is the outcome of the general impairment of brain function consequent on the increase of intracranial pressure; but unilateral defect of hearing, which, however, never amounts to complete deafness, may occur in the opposite ear when a tumour destroys the auditory centre in the first temporo-sphenoidal convolution, whereas when the hinder part of this convolution and the adjacent part of the second are affected, the variety of aphasia spoken of as word-deafness may result.

Reflexes.—The state of the plantar reflex and of the tendon jerks largely depends on whether the tumour is so situated as to interfere with the motor path or not. With such interference the plantar response is one of extension of toes instead of flexion, which is the normal response, and the tendon jerks are exaggerated. The greatest possible variations, however, occur in connection with the knee jerks, for even in the presence of hemiplegia they may be lost, and without such paralysis, exaggerated, diminished, or abolished knee jerks are met with, in addition to which they may be variable, disappearing and reappearing, it may be more than once, in the course of the case. It was at one time supposed that loss or vascillation of the knee jerk is suggestive of cerebellar tumours; but we are now aware that a tumour of the cerebrum, which is large, grows rapidly, or produces marked hydrocephalus, has a similar influence on the state of the knee jerks.

The *pulse* may be unaltered, or it may be slower than normal, in some cases markedly so; but irrespective of its previous behaviour, it may be much quickened as the end approaches, and may then also become irregular.

Respiration may be markedly affected by tumours in any situation, but especially by those in the neighbourhood of the *medulla oblongata.* Cheyne-Stokes' respiration may result, or what is by no means uncommon is a sudden cessation of respiration, which is the immediate cause of death in a considerable proportion of cases of cerebral tumour. *Hiccough* may be troublesome, but is not common.

The *temperature* is usually normal, or it may be even sub-normal, except where meningitis, pneumonia, bed-sores, or some such complication is responsible for its elevation; but tumours of the *medulla, pons, and basal ganglia* are sometimes attended *with hyperpyrexia.*

General Nutrition.—Although the patient's general nutrition may be good in the earlier stages of a cerebral tumour, there commonly results, sooner or later, a variable amount of emaciation, sometimes not more than is to be accounted for by vomiting, pain, want of sleep, or some complication; but in certain cases a marked degree of wasting that cannot be ascribed to any of these causes seems to depend directly on the tumour, or its effects on the brain.

Bed-sores.—The presence or absence of bed-sores largely depends on the care that can be bestowed on the patients.

Urine.—Polyuria, glycosuria, albuminuria, peptonuria, and phosphaturia all occur, though the last is alone common, while the other conditions have occasionally been met with, more especially in tumours of the fourth ventricle. Before ascribing any localising significance to such symptoms, the possibility of their being accidental accompaniments must be excluded.

DIAGNOSIS.—Three main questions have to be decided when a cerebral tumour is suspected: whether a tumour is present, the part of the brain in which it is situated, and its probable nature. The difficulty of solving these problems is inverse to the order in which they have been enumerated, for while it is generally possible to decide the first question, the second can much less frequently be determined, while a positive decision on the third is of necessity considerably more remote. The presence of headache, vomiting, and optic neuritis makes a tumour highly probable, but they are not conclusive proof of its existence, in that abscess, meningitis, cerebritis, hydrocephalus, anæmia, renal disease, lead poisoning, and certain other conditions may be attended by these three symptoms. Even if symptoms like convulsions and giddiness are present, we are left with possibilities other than tumour to account for the manifestations; when, however, focal or localising symptoms are added to those of general significance, diagnosis is much facilitated.

The exclusion of *Bright's disease* depends on the presence of casts as well as albumin in the urine, a high tension pulse, and hypertrophied heart. Examination of the fundi may further aid in the elucidation of the problem, but not necessarily so, in that retinal changes may occur with cerebral tumours which are closely similar to those seen in albuminuric retinitis, and papillitis without retinitis sometimes occurs in Bright's disease.

In profound *anæmia*, especially in young girls, the general symptoms of cerebral tumour may be present, in which case diagnosis depends on the absence of focal symptoms, the severe degree of anæmia present, and the fact that under treatment with iron, combined with good food and rest in bed, all the manifestations including the optic neuritis disappear. In some of these cases hypermetropia is also present, and its correction by suitable glasses results in cessation of the headache even before the other symptoms yield to the treatment directed to the anæmia. Ignorance of the fact that hypermetropia alone may give rise to changes in the discs resembling early papillitis may lead to an erroneous diagnosis of tumour; with suitable glasses, the headache and congestion of the discs disappear. More difficult than either of these groups of cases, however, is that in which young women with amenorrhœa, especially if there is little or no anæmia, present the general symptoms of cerebral tumour; indeed it may be quite impossible to arrive at a positive diagnosis. The exclusion of *lead poisoning* is usually not difficult, in that the symptoms of cerebral origin are usually preceded by other symptoms of saturnism. The patient's occupation or habits, a past history of attacks of colic, the presence of the blue line on the gums, wrist drop, and so on, serve to make the nature of the case clear.

Abscess.—In some cases of chronic encapsulated abscess it may be quite impossible to arrive at a correct diagnosis, but in general the following points have to be relied on for a differential diagnosis between abscess and tumour. It is of primary importance to exclude the causes of abscess, more especially the more common ones such as chronic disease of the ear and nose. Trauma while in favour of abscess can have no great weight, for, as we have said, blows to the head play an important, if obscure, part in the etiology of certain tumours of the brain. The mode of onset and progress of the symptoms may supply us with information, for while tumour usually develops slowly and pursues a uniformly progressive course, an abscess usually gives rise to serious cerebral symptoms which appear rapidly, and which may be followed by a latent period in which little or no manifestations are forthcoming. But on the other hand, some abscesses are insidious from the onset, progress slowly, and never give rise to characteristic signs. In any case where diagnosis is in doubt, the sudden occurrence of symptoms pointing to widespread meningitis or sinus phlebitis are naturally strongly in favour of abscess. With encapsulated abscess without concomitant meningitis, headache is less severe than in tumour, so too optic neuritis is both more frequent and more severe in tumour than in abscess. The absence of pyrexia does not exclude abscess, for without concomitant meningitis or sinus phlebitis, the temperature may not only be normal but even subnormal throughout the whole course of an abscess. The position of the lesion, if indicated, is of some value in diagnosis, for in the vast majority of cases abscesses are either in the tempero-sphenoidal lobe or cerebellum, so that where the symptoms point to some other region of the brain as the part affected, the probabilities are much in favour of tumour.

The difficulty of distinguishing *meningitis* is proportional to the chronicity of the process, and, more especially to the degree of its limitation. A localised syphilitic meningitis may give rise to symptoms which are indistinguishable from those that result from tumour; but the only difficulty likely to arise with regard to acute meningitis is that of deciding whether a tumour co-exists in the tuberculous variety. A history of headache and vomiting for some time before the more acute symptoms became manifest is in favour of tumour, as is severe optic neuritis with much swelling of the disc, especially if the condition is detected soon after the apparent commencement of the meningitis. Chronic cerebritis may give rise to symptoms identical with those that result from tumour, as may internal hydrocephalus; but the former class of case is rare, and the latter only exceptionally causes difficulty in diagnosis.

When symptoms occur suddenly with cerebral tumours, especially hemiplegia, the question is raised as to whether a vascular lesion is alone responsible for the occurrence; but careful inquiry will usually elicit a history of pre-existing symptoms of slow development, and the detection of optic neuritis would further tend to make the nature of the case clear. Cases of tumour, in which giddiness, vomiting, and deafness are prominent early symptoms, may for a time, at any rate, be mistaken for *Menière's disease*, but diagnosis does not usually long remain in doubt. As has been said, the mental state in some cases of cerebral tumour resembles that of *general paralysis of the insane*, and the character of the speech may make the resemblance still greater; but headache, vomiting, and optic neuritis, if present, indicate tumour. When jerky inco-ordination is a symptom, the case may be erroneously regarded as one of *disseminated sclerosis*, but in the latter disease the tremor usually affects both sides as opposed to one in tumour, nystagmus is common, and optic neuritis exceptional, and when present usually slight; atrophic pallor of the discs, on the other hand, is common. Moreover, headache and vomiting are very much less commonly met with than in tumour. Those cases of tumour where general convulsions are the only symptoms for years, naturally cannot be diagnosed from *idiopathic epilepsy* until optic neuritis or some other sign of the gross lesion is forthcoming: but the opposite mistake of supposing that fits having a local beginning of necessity mean a gross lesion, must be guarded against. *Hysterical manifestations* may so mask the organic affection as to lead to errors in diagnosis, but this will usually be avoided by a determination never to diagnose hysteria as the sole condition present, until every possible source of organic affection has been negatived. In the search for some sign of organic disease, optic neuritis or some other defect of like significance will generally be found. It is less likely that phenomena that are purely hysterical will be mistaken for the effects of an intracranial tumour, for due regard to the special features which characterise hysterical manifestations in general as opposed to those of organic origin should, as a rule, prevent this. Moreover, there are important distinctions even where the phenomena appear similar, in which connection the grouping of the symptoms may be of especial value.

A knowledge of regional diagnosis of cerebral[1] disease in general together with what has been said in this connection when the symptomatology of intracranial tumours was under consideration, must suffice as regards the information on which we depend for the localisation of a tumour. Many factors serve to make the problem a very difficult one. Apart from one of the "silent regions" of the brain being affected, the marvellous possibilities of compensation of which the nervous system is capable, result in a singular absence of localising symptoms in many cases. One of the greatest bugbears to localisation, however, is the general increase of intracranial pressure, which so distorts the clinical picture as to make accurate localisation an impossibility in a large proportion of cases. The instances in which the effects of vascular occlusion occur at some part remote from the seat of tumour are met with too infrequently to make this possibility a serious stumbling-block in localisation.

CORTICAL AND SUB-CORTICAL TUMOURS.—Tumours near the surface of the brain afford the best chances of successful surgical intervention, so that it is important to recognise the special features which justify the localisation of a neoplasm in or just beneath the cortex. It is when a tumour is in, or contiguous to, the sensori-motor cortex of the Rolandic region of the brain that the most definite indications are supplied. Paralytic phenomena are of much greater value in localisation than are those the result of over-action, for when there is paralysis the disease must involve the nerve centres concerned with the affected part, while convulsion may depend on irritation of some contiguous part. The special feature of paralysis of cortical origin is that it is limited in range, for, owing to the wide expanse of area on the surface of the brain, partial paralysis, such as a monoplegia, is much more commonly met with here than where the motor fibres become collected within narrower limits in deeper parts of the brain. The lips and tongue are affected when the lowest part of the motor region is involved, the face when the part just above this is implicated; the arm suffers most when the middle region of the Rolandic area is the seat of disease, while the leg is involved when the morbid process abuts on the longitudinal fissure. If the paralysis is entirely limited to the leg, then the probabilities are that the paracentral lobule is the seat of disease. The local commencement of a convulsion is the characteristic feature of tumour of the cerebral cortex, for such a mode of commencement is exceptional if the disease is situated elsewhere. The part in which such convulsions commence may indicate the seat of lesion, but when a destructive lesion is present the paralysis indicates the precise seat of the tumour, while the convulsions originate in parts contiguous to this. Moreover, some parts of the cortex appear to be more prone to discharge than others, so that even with a tumour situated at some distance from them they may, nevertheless, be the first centres to discharge, as is the case in regard to the centres which are concerned

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[1] For "Cerebellar Tumour" see "Cerebellum."

with the movement of the head and eyes to one side, and those which have to do with the movements of the thumb and fingers. This being the case, the evidence obtained from convulsions has to be discounted when the discharge begins in any such highly sensitive centres. Convulsions which begin locally may remain limited to the part first in a state of spasm, or they may spread so as to involve the whole of the side, or may subsequently become bilateral. Instead of the cortical discharge manifesting itself by muscular spasm, it may only engender a subjective sensation of some kind which may spread up a limb and ultimately involve the whole of one side of the patient before any convulsions appear. Such sensory discharges are as good evidence of the seat of lesion in the centres which are concerned with the movements of the limb as if the initial phenomenon had been a muscular spasm. In some cases one of the special sense centres may be the seat of discharge, in which case the evidence thus supplied as to the locality of the tumour is that to be relied on rather than any information derived from any subsequent convulsions that may result. There may be loss of sense of position in the paralysed limbs, but this is not of any very great value as a sign of disease of the cortex, in that it also occurs when the lesion is in the internal capsule. Common sensibility is affected in a way that it is important to recognise. There is slight blunting to tactile impressions in the parts paralysed, and the apex of the limb suffers in greater degree than do the proximal segments; moreover, patients the subjects of such defect refer erroneously impressions which they do perceive, so that a touch on the backs of the fingers is perhaps referred to the back of the hand, while one on the latter situation is referred to some part of the forearm.

Crura Cerebri.—The evidence on which we rely for the localisation of a tumour in the crus is alternate hemiplegia characterised by affection of the limbs, and it may be the lower part of the face on the opposite side, while the muscles supplied by the third nerve are paralysed on the side of the lesion. When the third nerve is completely paralysed there is ptosis, external strabismus, and mydriasis; but there may be only a partial affection of the nerve, in which case the levator palpebrae and the internal rectus are most liable to be affected. Both third nerves may suffer in consequence of involvement of both crura by the growth, in which case the limbs on both sides may be affected. Instead of paralysis, the motor defect in the limbs in some cases is ataxic, while in others tremor has been present. Anæsthesia of the opposite side of the body does not usually result when a tumour involves the tegmentum, no doubt because new growths tend so much to displace rather than destroy the nerve elements.

Pons.—The characteristic sign of a pontine lesion is alternate paralysis, in which the limbs are affected on the opposite side and the parts supplied by the fifth, sixth, or seventh cranial nerves on the same side as the tumour. Affection of the auditory nerve is exceptional. The most common form of these alternate hemiplegias is that in which there is facial paralysis of peripheral type on the side of the lesion and paralysis of the limbs on the opposite side; but in order to bring about this condition of things the tumour must be situated in the lower half of the pons, so as to involve the nucleus or the roots of the facial nerve as they emerge from the nucleus. When the roots of the sixth nerve are affected, there is paralysis of the external rectus on the same side, with inability to turn the eye out; but if the sixth nucleus is involved, there is in addition affection of the internal rectus of the opposite eye, in so far as it is concerned in the act of conjugate turning of the eyes to the side of the lesion. In the paralysis which results from such a lesion, the eyes may be turned slightly to the opposite side, but in any case the patient is unable to turn them to the side of the lesion. Although affected in this way, the internal rectus usually retains its power of acting with its fellow on convergence. When a tumour irritates instead of destroying the sixth nucleus, spasm of the muscles may turn the eyes towards the side of the lesion. In the event of the fifth nerve being affected, its sensory portion is more liable to suffer than is its motor division, but not uncommonly both are affected, so that there results anæsthesia of the face on the same side; it may be preceded by neuralgic pains of similar distribution, and paralysis with atrophy of the muscles of mastication on the same side. In so far as the paralysed limbs may be anæsthetic, in some cases there may be a crossed hemianæthesia in which the face is affected on the side of the lesion and the limbs on the opposite side. Bilateral symptoms frequently occur as a result of tumours in the pons, either owing to secondary involvement by pressure on, or extension to, the opposite side of the organ when the growth is originally unilateral, or owing to the neoplasm commencing near the middle line. In such cases there may be bilateral paralysis of the limbs and tongue in addition to similar implication of the fifth, sixth, and seventh nerves on both sides. The most varied pictures may, however, result from pontine lesions, and ataxic disorders have been met with in connection with growths in this situation.

MEDULLA OBLONGATA.—The features by which tumours in this region are to be recognised are defects consequent on implication of the glosso-pharyngeal, vagus, spinal accessory, or hypoglossal nerves, together with, it may be, paralytic defects in the limbs on both or only on one side. It thus happens that in cases of this kind defects of articulation, difficulty of deglutition, disturbances of the heart's action and of respiration, unilateral or bilateral paralysis of the palate with loss of reflex action, and paralysis of one or both vocal cords are met with. As in the case of the crus and pons, the motor disturbance in the limbs in some cases is due to inco-ordination rather than to true paralysis.

Although it is only in a comparatively small number of cases that an opinion can be expressed as to the nature of the neoplasm with anything approaching to certainty, it is nevertheless important that an attempt should be made to decide this question in every case owing to the important bearings that the nature of the tumour has both on prognosis and treatment. *Syphilis* supplies the largest number of tumours that can be recognised with the greatest degree of probability, while to *tubercle* may be assigned the second place in this connection. As regards other forms of intracranial tumour, it is largely a matter of surmise, except when a primary growth of known nature exists, or has been removed from some other part of the body, in which case an almost positive opinion becomes possible both in regard to carcinoma and sarcoma. So too in the case of parasitic cysts, the nature of that inside the skull may be made evident by the information supplied by one or more in other parts of the body. The points in favour of a syphilitic growth are the age of the patient, a history of infection or of congenital origin, and, in the absence of these, the detection of associated lesions of known syphilitic nature. Evidence that the tumour is on the surface of the brain is in favour of its being syphilitic, whereas it is less likely to be of this character if deeply seated in a cerebral hemisphere, and still less likely if it is in the cerebellum. So too it is unlikely that a tumour which is growing very slowly is of this nature. The best evidence is, however, supplied by *treatment*, for the symptoms produced by no other form of tumour can be so completely removed by drugs as is possible in syphilomata. As regards evidence of tuberculous tumours, the detection of disease of a like nature in some other part of the body is all important, less so in children, however, than in adults, for in the latter, as we have already said, intracranial tumour of this nature rarely exists without concomitant tuberculous disease of the lungs. In the absence of such evidence a scrofulous diathesis, or hereditary history of tuberculosis must be allowed to exert some influence on our deliberations. The fact that the patient is a child, and that the growth is in the cerebellum in a patient at this time of life, are also of some value. Evidence of rapid growth at first, followed by a stationary period, is suggestive, while when symptoms of tumour become complicated by those of general meningitis the probabilities in favour of tubercle become very strong. Treatment is only of secondary value in aiding to elucidate the problem, though when other indications suggest tubercle improvement under tonics may be allowed some weight in diagnosis. We possess little information that can aid us in determining the nature of other forms of growth, though proof that a tumour originated outside the brain is in favour of *sarcoma*, while evidence of very slow growth is in favour of *fibroma* or *glioma*, though the latter may be one of the most rapidly growing forms of neoplasm. Sudden symptoms indicative of hæmorrhage into the tumour is also in favour of glioma.

Prognosis varies greatly in different cases, though in no instance can a cerebral tumour be regarded otherwise than as serious. Several considerations influence prognosis, paramount among which is the nature of the growth. If syphilitic, there is a good chance that it may be satisfactorily removed by drugs, but it must be remembered that on the one hand such growths may recur, and that on the other absorption of the tumour does not of necessity mean disappearance of all the symptoms to which it gave rise, for irreparable damage may have been done to the nerve elements at the seat of growth, or the resulting scar tissue may cause persistent epilepsy, though the latter class of case may prove amenable to surgical intervention. Then again syphilitic disease of the cerebral vessels may be so advanced that absorption of the tumour avails the patient little. Tuberculous tumours are not uncommonly arrested, but in that disease of a like nature may coexist in some other part of the body, prognosis as regards life may turn on, for instance, the pulmonary condition rather than on the cerebral. In the case of such a tumour that has become stationary, the symptoms to which it formerly gave rise may improve greatly or may entirely disappear although the tumour is still present, notably is this the case when the cerebellum is its seat. In all other forms of cerebral tumour prognosis is grave, except where there is a reasonable hope that the growth can be successfully removed by operation. In any case of intracranial tumour death may be unexpectedly sudden, but apart from this accident the probable duration of life has to be estimated from the history of the length of time that symptoms had existed, the rate at which they are progressing, and the degree of their severity; the more urgent the symptoms the more grave is the immediate outlook. The seat of the tumour has also important bearings on this question, for those which involve the medulla oblongata or pons as a rule terminate life more rapidly than tumours in other situations. Valuable information may be afforded by the state of the optic papilla, for intense double optic neuritis of rapid onset is of serious import, while when there is evidence that the neuritis is very chronic the probabilities are that the unfavourable progress of the disease will be slow. In the absence of trephining, one of the most favourable signs *in prognosis* is a diminishing optic neuritis.

Treatment.—The treatment of cerebral tumours is of two kinds, curative and palliative; but unfortunately the cases in which cure is possible are far outweighed by those in which palliative measures can alone be adopted. In either form of treatment we have both drugs and surgical procedures which are of use. Two drugs are alone of any real value in the curative treatment of cerebral tumours, *iodide of potassium* and *mercury*, and unfortunately syphilitic tumours alone result in cure under their influence. Treatment by these drugs does a certain amount of good in tuberculous tumours also, and temporary improvement may result in other forms of intracranial growth, so that improvement under this regime does not necessarily mean that the tumour is syphilitic, although in this variety alone can cure be looked for under such treatment. Even syphilitic tumours are not, however, always completely cured by these drugs, for a certain amount of thickening and scar tissue not unfrequently remain which no amount of drugs can remove. The meninges are adherent to the cortex under such circumstances, and the irritation which results may lead to persistent epilepsy, followed, it may be, by dementia and insanity. Thus even in syphilitic cases when drugs do not succeed in completely removing the disease, as evidenced by a continuance of some or all of its former manifestations, the cases form a proper and suitable group for surgical intervention, provided the position of the lesion is one accessible to operation, and there is not already evidence of widespread syphilitic disease of the cerebral arteries. The temporary improvement that may follow the use of iodide of potassium and mercury in cerebral tumours other than those of syphilitic nature is unfortunate, in that it engenders false hopes, and occasions an amount of delay that might otherwise not be entertained where surgical intervention is under consideration. To be of real value in the treatment of syphilitic tumours of the brain iodide of potassium must be given in full doses, such as thirty grains three times a day, and the drug ought to be pushed beyond these limits before it is abandoned in any case of the kind in which it does not appear to be influencing the condition. Moreover, when iodide of potassium alone fails in its object a beneficial result may, nevertheless, be obtained by the administration of mercury in conjunction with the other drug. Inunction of the mercury or its administration in the form of gray powder are preferable to other modes of exhibiting the drug in these cases. *Cod liver oil* and *tonics*, such as are in vogue in the treatment of tuberculous disease elsewhere, are also of value in the treatment of tuberculous tumours of the brain. Apart from the temporary improvement observed under treatment by iodide of potassium in some cases none of the other forms of cerebral tumour are favourably influenced by drugs. Even arsenic, which has been tried in cases of sarcoma, owing to the restraining influence that this drug appears to exert on this form of growth in other parts of the body, has had no obvious influence.

The curative treatment of cerebral tumours by surgical means has met with brilliant success in the hands of skilled surgeons of special experience; but only a comparatively small proportion of cases of the kind allow of surgical intervention with a view to enucleate the growth, a state of things due to a variety of circumstances. A large number of tumours are so situated as to be beyond the reach of the surgeon, others though accessible occupy too extensive an area of the brain, or are of such an infiltrating nature that they cannot be completely removed.

Where the cure of a cerebral tumour is impossible much may yet be done to alleviate the patient's suffering. All physical exertion should be avoided, and as much mental rest as possible secured; alcohol should be forbidden, and constipation guarded against. Of the various drugs that are in use for the relief of the pain phenacetin, antipyrin, croton chloral, cannabis indica, and morphia are the most serviceable, though the last named should be only given when the paroxysms of pain are so severe that none of the other drugs give relief. Cold to the head in the shape of the ice-bag is of great service, and is to be preferred to either counter-irritation or leeching. Convulsions are best controlled by bromides and chloral. Where medicinal treatment fails to relieve the urgent symptoms of a cerebral tumour, we have in trephining a measure that is of the greatest possible benefit in suitable cases. Not only are headache, vomiting, and, it may be, convulsions relieved, but impending death may be averted by the relief of intracranial pressure, and, in any case, life is prolonged, and the time left to the patient to live made infinitely more tolerable than without the operation. Another important consideration is the influence which trephining has on the optic neuritis, which quickly subsides and may completely disappear, so that, provided post-neuritic atrophy is not in progress, sight may thus be saved.

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Brain—Atrophy, Hypertrophy, Cysts, Inflammations.

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Atrophy of the Brain

Atrophy of the brain may be general or local. A limited degree of general atrophy is a senile change, where shrinkage of brain matter is not infrequently associated with a proportionate increase in the amount of cerebro-spinal fluid. Apart from this, in some rare cases atrophy of the whole brain occurs, although much more frequently the change is local.

Pathological Anatomy.—There is always an increase of connective tissue at the expense of nerve-cells, and the result may be a softer or a harder brain substance, depending upon the kind of sclerosis which accompanies or is the result of the atrophic process.

Etiology.—Atrophy may occur: (1) Before birth, as the result of maternal injury or imperfect development of the embryonic structures.

(2) At birth, as the result of injury to the skull, hæmorrhage causing pressure, or possibly meningeal inflammation.

(3) During the first years of life, when atrophy, often partial, is due to defective development, gradually causing more and more definite alterations in the brain structure.

Partial atrophy, while it may affect any part of the brain, more generally occurs in one cerebral hemisphere in whole or part, in the cerebellum, either one or both lateral lobes, or in the pons.

There may be atrophy of one part of the brain, consequent on, or associated with, a similar condition in another part, as, for example, one cerebral hemisphere and the opposite hemisphere of the cerebellum, or one cerebellar hemisphere and the opposite basal ganglia and olivary body.

Clinical Features.—While it is unnecessary to refer here to the symptomology of hereditary cerebellar ataxia, or other more or less defined types of disease associated with atrophy of the brain, it is sufficient to remark that intellectual feebleness is generally coincident with imperfect development. Hemiplegia or unilateral weakness, very frequently athetoid movements such as post-hemiplegic chorea, inco-ordination, and not uncommonly epilepsy, are associated with a greater or less degree of idiocy.

Occasionally no symptom has resulted in cases where well-marked atrophy existed, but these are both rare and inexplicable.

No special *treatment* can be given other than that based on general principles.

Hypertrophy of the Brain

This is a subject upon which much discussion has taken place, and many theories have been formulated since Virchow's paper in 1856.

Hydrocephalus and neoplasms are not included under this head, and it is necessary in many cases carefully to distinguish between hydrocephalus and the condition we are considering. Hypertrophy may be local, but is more frequently general.

Pathological Anatomy.—Hypertrophy of the brain, as Gowers points out, should mean proportionate increase in all the constituent parts of which the organ is normally composed. There are many instances of large brains in persons specially endowed with genius and intellectual power, and also in those who are mentally deficient. In the former Gowers' definition holds true, but in the latter there is probably an increase of neuroglia, as Virchow stated, in excess of nerve-cells and nerve-fibres.

There is generally a proportionately greater amount of white matter, and the consistency of the brain is much firmer than normal. In sharp contrast to hydrocephalus there is a diminution in amount of cerebro-spinal fluid, and the brain is anæmic, probably due to increased pressure.

Etiology.—It is possible that deficient resisting power of the cranium may account for the increasing growth of the brain substance, and therefore the greater number of cases occur before the sutures of the skull have ossified, or where ossification is greatly delayed, as in rickets.

It is not surprising to find that the condition may run in families for several generations.

Clinical Features.—Many symptoms, such as mental enfeeblement, idiocy, fits, headache, mental excitement, followed by temporary coma, have been ascribed to this condition; but, as Gowers rightly points out, they may or may not be associated with it, and it is in his opinion incorrect to place the hypertrophy of brain substance in a causal relationship to the very varying symptoms. Still, many later observers agree in associating these groups of symptoms with the anatomical or pathological condition—hypertrophy of the brain,—and give many convincing instances in support of their arguments.

Some cases resemble hydrocephalus, others meningitis, and others again merely a greater or less degree of mental apathy, with slow movements as in eating or drinking, and still more lethargic thought and speech.

In these cases the balance of neuron and neuroglia is certainly not normal, and therefore they may not be accurately described by the term hypertrophy, but rather by enlargement of brain, with a greater or less degree of sclerosis.

The treatment is as unsatisfactory as the *prognosis* in all cases where the hypertrophy has produced any abnormal phenomena.

CYSTS OF THE BRAIN AND CYSTIC DEGENERATION

Cysts may be the result of: (1) Defective development (porencephaly); (2) cysts formed as the result of an old hæmorrhage; (3) cysts representing an old patch of softening; (4) cysts forming in the substance of encephalic tumours; and lastly, (5) dermoid and hydatid cysts.

(1) *Porencephaly.*—This term ought, as Heschl suggested, to be solely used for cavities due to embryonic errors, where, from absence of brain substance (cortex and subcortical tissue), a cavity is left which in most cases communicates with the ventricle. Cavities formed as the result of hæmorrhage or from softening following on embolism or thrombosis should not be included under this title; still, as Sachs suggested that where, as the result of interference with the fœtal brain, a cavity resulted, such cases might properly be called acquired porencephaly. In some cases of porencephaly the cavity is entirely covered in by brain substance, in others it is invested by pia and arachnoid, or the pia may be reflected over the walls of the cavity, especially near the cortex, and the arachnoid may roof in the funnel-shaped space. The cerebral hemispheres and the island of Reil are common sites for such cavities. A radiating distribution of the

cerebral convolutions around the cavity has been described by Kundrat as typical of true porencephaly, and the primary fissures of the brain are always represented. When the cavity is not superficial, the cerebral convolutions situated over it are narrowed and depressed. Kundrat believes that whereas in true cases the pia either closely covers the cortex when the cavity is subcortical, or partially lines the cavity where the cavity communicates with the subarachnoid space, in acquired or spurious cases the pia neither closely covers the cortex nor dips into the cavity in the same way as in the true cases. Where the cavity communicates with a lateral ventricle, the ventricle is dilated and its ependyma thickened. The skull in these cases often undergoes alteration in shape, the affected side being in some cases increased, in others diminished in size, and the face may be correspondingly asymmetrical.

The clinical features vary greatly with the size and site of the cavity. There is almost always mental disturbance, and often idiocy. Loss of speech, sometimes sight and hearing, are not infrequent, as in a case seen by the author, while spastic paralysis with contracture, athetosis and great modification in size and shape of limbs are common, while epilepsy often occurs. Hemiplegia is not infrequent.

The diagnosis of the existence of a cavity may be difficult, although it may be suspected from the group of symptoms present.

The prognosis is good, so far as life is concerned, for at least some years but recovery is impossible.

The treatment must obviously be based only on general principles.

(2) Cysts resulting from hæmorrhage generally occur in persons who survive a cerebral hæmorrhage for some months. The walls are firm, ochre coloured, and contain hæmatoidin and hæmosiderin crystals, while the contents vary with the age of the cyst.

(3) Cysts resulting from a patch of softening contain the fluid remains of softened brain tissue from which the fat and solid elements have been removed.

(4) Cysts are not infrequently met with in sarcomata, sometimes in other tumours, and require no special comment.

(5) Dermoid cysts are exceptionally rare. Kruse has described one case, and has noted two previous references to cases described in literature. In Kruse's case the cyst was situated in the neighbourhood of the fourth ventricle, and had caused no symptoms; in one of the others the cyst was in the cerebellum, and was associated with paraplegia.

(6) Hydatid cysts.—A number of cases of cerebral hydatid cysts have been described. The symptoms, mainly irritation followed by depressive phenomena, closely resemble those resulting from intracranial tumour. The cysts are generally localised in position, but may be multiple, and very frequently become superficial. They may dry up, and so lead to recovery without any interference.

Cystic degeneration is a disseminated collection of cysts, many barely visible to the naked eye, and the larger ones being about the size of a pea. These may run together, and they are believed by most authorities to be due to dilatation of perivascular lymph spaces, although the evidence is not conclusive. They have been met with in cases of locomotor ataxia, progressive paralysis, and also in cases with no mental or other signs of brain disturbances.

CHRONIC PROGRESSIVE SOFTENING OF THE BRAIN

In the white matter of the cerebral hemisphere there is a region which is supplied partly by cortical and partly by central nutrient arteries, and which therefore suffers when, as the result of any arterial disease, there is deficient nourishment conveyed to the brain. Here, as the result of atheroma, for example, patches of softening occur in late middle life or old age.

The group of conditions described under chronic progressive softening may not all be due to vascular disease, but those not so included are very inexplicable. The softening is slow in development and progressive in advance, and may eventually involve an extensive area of brain substance.

The white matter more frequently suffers in the cerebral hemispheres, while in the cerebellum it is generally the gray cortical portion.

Pathological Anatomy.—The affected areas are generally whitish in colour, and of soft consistence. They are composed of the remains of degenerated nerve-fibres, and considerable atrophy of brain substance may follow as a result. The vessels in the majority of cases are certainly diseased.

The etiology is probably almost entirely that of vascular disease, especially atheroma, and the condition is one often affecting debilitated patients who have passed middle life.

The clinical features, as far as they can be described from recorded cases, are very similar to those met with in cerebral thrombosis, only more gradual in onset and advance.

There may be premonitory symptoms, such as headache, giddiness, and mental enfeeblement, followed by unilateral sensory and motor phenomena, such as tingling, numbness, weakness in one limb, and spreading to the other parts of the same side. Spasm may occur, but is infrequent, and mental weakness, although eventually generally well marked, may at first not be recognisable. The disease lasts for months, or even years, and during this time the symptoms become more marked, death being due sometimes to bed-sores, more often to intercurrent disease.

The diagnosis is often difficult. It is closely allied in symptomatology to cerebral thrombosis, and it also resembles, according to Gowers, intracranial tumour. It certainly lacks the definiteness of either of these. Optic neuritis, severe headache, vomiting, and focal symptoms suggest tumour.

The prognosis is against recovery, although the patient may remain for months in *statu quo.*

The treatment much resembles that usually recommended for cerebral thrombosis, but is not very hopeful.

ACUTE ENCEPHALITIS

Inflammation of the brain may be the result of—

(1) Inflammation of the membranes, to which it is a necessary sequel.

(2) Injury to the brain, such as by a blow or fracture, and here meningitis is not an infrequent accompaniment.

(3) A local irritant, such as disease of a cranial bone, the pressure of a tumour, and so forth.

(4) A patch of cerebral softening or a hæmorrhage, the degree of inflammation varying greatly in different cases.

(5) The presence of a pus-forming or infective organism.

(6) Closely allied to the last, there are cases which are not all proved to be due to the local presence of organisms, but which occur in infective diseases, such as syphilis, influenza, typhoid fever, erysipelas, scarlet fever. These cases are often described as disseminated encephalitis, or diffuse non-suppurative encephalitis.

(7) In tetanus, in hyperpyrexia, and in not a few cases of toxæmia intense local areas of hyperæmia, with sometimes hæmorrhages, and not

infrequently perivascular aggregation of leucocytes, are often seen in the medulla, phenomena strongly suggesting an inflammatory process.

It should be stated that purulent encephalitis is described under "Cerebral Abscess," and is not included here (p. 92).

Pathological Anatomy.—The area affected is always red in colour, thus sharply distinguishing cerebritis from a patch of ordinary necrotic softening. This is due to hæmorrhage into the area affected and distension of vessels, especially capillaries, and there are always numerous leucocytes, especially around the vessels. The nerve-cells and fibres undergo degeneration, the cells swelling up, losing their processes, often becoming pigmented, and eventually may break down, the medullated fibres undergoing Wallerian degeneration, and the axis cylinders forming bead-like swellings before they too become unrecognisable. There may be well-marked proliferation of neuroglia cells in certain cases.

The area of cerebritis is never sharply outlined from healthy tissue, but neighbouring brain matter suffers definite injury. Large areas or small areas may suffer. There are cases which have been called acute focal encephalitis, and most of these are hæmorrhagic. Strümpell contended for the recognition of this condition, which he believes to be analogous to acute poliomyelitis anterior, and Moebius, Schmidt, and others have described such cases as occurring in adults and children. In one typical case an area of deeply blood-stained debris was found in the left corpus striatum, and minute hæmorrhages were scattered over other parts of the brain. The lesion differs from that found in the cord, inasmuch as the inflammation is not limited as definitely to the gray matter. Lamy, Redlick, and Beyer describe polio-encephalitis and poliomyelitis anterior acuta occurring in the same patients. Wernicke first drew attention to a polio-encephalitis acuta hæmorrhagica superior where the area of cerebritis involves the floor of the fourth ventricle and the aqueduct of Sylvius. This is simply an acute focal encephalitis in which hæmorrhage is marked, and which occurs in a special site. Where recovery occurs, partial absorption of the softened area may result, just as in an area of non-inflammatory softening, or before a fatal issue suppuration may ensue.

In disseminated encephalitis there may be little scattered foci of inflammation, sometimes containing micrococci or other organisms, and in syphilitics, scattered areas of sclerosis have been described by Charcot and Gombault, which are more often chronic than acute. Secondary degeneration may result from the destruction of brain tissue.

The etiology has been already to some extent given under the table at the commencement of the article. Acute focal encephalitis has resulted from influenza, scarlet fever, measles (Henoch), erysipelas, diphtheria, while lately whooping-cough (Neurath) and mumps (Putnam) have been held responsible for individual instances.

Acute polio-encephalitis hæmorrhagica superior appears to be more specially the result of alcoholism, sulphurous acid, and various poisons, such as from decomposing meat, and lastly influenza.

The clinical features vary greatly. In a simple case of acute cerebritis, headache, sometimes vomiting, convulsions, delirium, but rarely optic neuritis, might be described as general phenomena, apart from any focal symptoms likely to result.

In acute focal encephalitis or polio-encephalitis acuta (hæmorrhagica), the disease occurs in young children, is acute in onset, and accompanied by fever, vomiting, headache, vertigo, convulsions, rarely general, and sometimes bending backwards of the head. There is generally a well-defined stage of irritation, which gives place to a paralytic stage. The symptoms just referred to characterise the irritation stage, while paralysis of muscles or limbs, monoplegia, hemiplegia, sometimes aphasia, with eventually stupor and coma, develop during the second part of the illness. Cheyne-Stokes' breathing is not uncommon, and the pulse is often rapid. Optic neuritis may occur, and sometimes paralysis of cranial nerves. Death generally results in one to two weeks. Where recovery ensues, such sequelæ as epilepsy, hemichorea, and often imbecility, very frequently develop. In polio-encephalitis acuta hæmorrhagica superior, besides the ophthalmoplegia to which it gives rise, there is the same sudden onset, generally in a patient who has long indulged in excess of alcohol. There is generally little pyrexia, but sleeplessness, restlessness, almost amounting to delirium, rarely stupor, and then the gradual increasing ophthalmoplegia, with sometimes the levator palpebræ superioris or the sphincter iridis exempted. Nystagmus is common, and often difficulty in walking, much like cerebellar ataxia, is noted. Optic neuritis may also occur in some cases.

In what is described as acute diffuse non-suppurative encephalitis, the lesion may closely resemble in character the hæmorrhagic polio-encephalitis already referred to, being in this instance simply more extensive in area; but there are other cases which are rarely so acute, and if they begin acutely, generally become chronic. These are probably almost entirely of syphilitic origin, although any toxic agent may be responsible for a proportion of the cases. The clinical features of this type of encephalitis, without entering upon the subject of cerebral syphilis, consist of dull, sometimes deep-seated, headache, fever with delirium, or occasionally stupor. Convulsions and focal phenomena, such as twitching, spasms, and later paralysis, may be well marked, and not infrequently there is anæsthesia. The disease may advance fairly rapidly, or may become quiescent and more chronic. In some cases an extensive area of brain, such as one hemisphere, may be involved.

So-called disseminated encephalitis may give rise both to general and focal symptoms, and may eventually become, where pus-forming organisms are present, a disseminated suppurative encephalitis.

No special reference is required to the clinical features due to the inflammation resulting from a hæmorrhage or patch of softening (non-inflammatory in origin). It is the irritation of a foreign body, and probably the rise of temperature, twitchings, and irritation phenomena manifested twelve to twenty-four hours after a cerebral hæmorrhage are due to this frequently trivial and very localised encephalitis.

The Diagnosis.—It is always difficult to be certain where encephalitis has commenced until definite symptoms referable to the brain render the diagnosis certain. This is so, for instance, in traumatic cases where the brain has been injured by a punctured wound.

The extraordinary acuteness of the hæmorrhagic cases and the ophthalmoplegia where polio-encephalitis acuta hæmorrhagica superior occurs help greatly in the diagnosis. In the diffuse non-suppurative encephalitis due to syphilis there may be the greatest difficulty in recognising the exact nature of the disease, and distinguishing it from diffuse meningitis. The two conditions are often combined, but as a rule diffuse encephalitis, if recovered from, leaves marked permanent effects, and in diffuse encephalitis there is more intellectual loss than in diffuse meningitis. A focal encephalitis may, if recovered from, leave no mental defect. In all the forms of non-suppurative encephalitis, optic neuritis is much less common than in cases of cerebral tumour or abscess, but it does occur.

The prognosis varies with the type of encephalitis under consideration. It is bad in the hæmorrhagic types, and in all cases where a virulent toxin or micro-organism is concerned locally with the condition. Syphilitic cases may improve under treatment, and the inflammation around a hæmorrhage or patch of softening is only serious when it is very severe in degree. The encephalitis associated with traumatic, tuberculous, or other forms of meningitis may be considered to be secondary to the meningeal inflammation, but if very acute, is inevitably fatal.

The treatment is much the same as for meningitis: rest, sedatives, cold, or counter-irritation to the head, purgation, and especially the use of calomel. For very acute cases the sick-room should be kept darkened, ice bags to the head, and bleeding or leeching is of service unless in very anæmic patients. Antipyrin, salicylates, and quinine have been recommended, and also large doses of calomel.

Antisyphilitic remedies should be ordered in suitable cases, and such sedatives as bromides, hyoscine, and other opiates are frequently useful.

Chronic Encephalitis

Any of the acute forms described already may become chronic, and as the membranes often suffer along with the brain, the term meningo-encephalitis has sometimes been used, and especially for the more chronic forms. There are probably many forms of encephalitis which are chronic, such as the encephalitis found in the neighbourhood of tumours, and it is possible that in some cases of cerebral sclerosis there may be an inflammatory element in the patches so affected. Where brain substance has been compressed by a hæmorrhage it undergoes hardening, and the connective tissue in it increases in amount at the expense of nerve-elements proper, and this has been thought to be a chronic inflammatory process, although it is very doubtful. There is no reason to doubt that sclerotic patches, such as may occur, although rarely, in syphilis, are of inflammatory origin, and are in fact a chronic encephalitis. Such cases have been described by Charcot, Gombault, and others.

In general paralysis of the insane there is a chronic inflammation of the membranes, including the ependyma of the ventricle, with, in addition, a superficial chronic encephalitis. The *clinical features* require no special reference here, and the *treatment* must be on general lines.

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Brain, Surgery of.

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In dealing with cases of disease or injury to the brain it is necessary not only to diagnose the nature of the disease or injury, but also its exact situation. Unfortunately there is still a considerable part of the brain the exact functions of which are not understood, and this remark applies particularly to the right hemisphere; but of late years, owing to the researches of Ferrier and others, we have become cognisant of the relation between certain *areas* of the brain and the parts of the body which they control.

These areas have been mapped out on the brain by experimental and clinical research, and as the result of *post-mortem* observation, and do not necessarily correspond exactly with definite anatomical convolutions. It should be understood that the representation of any set of muscles, *e.g.* of the muscles of the thumb, is not strictly confined to the *area* allotted to it, but is only centred or focussed in the *area*, and spreads into other surrounding parts of the brain, fading gradually away as you leave the focus area. This explains the fact, that if the area corresponding to any set of muscles have been destroyed by disease or removed by operation, and paralysis of these muscles thereby caused, it is possible for the functions of the removed area to be gradually taken up, more or less completely, by surrounding parts of the brain, and power to be restored.

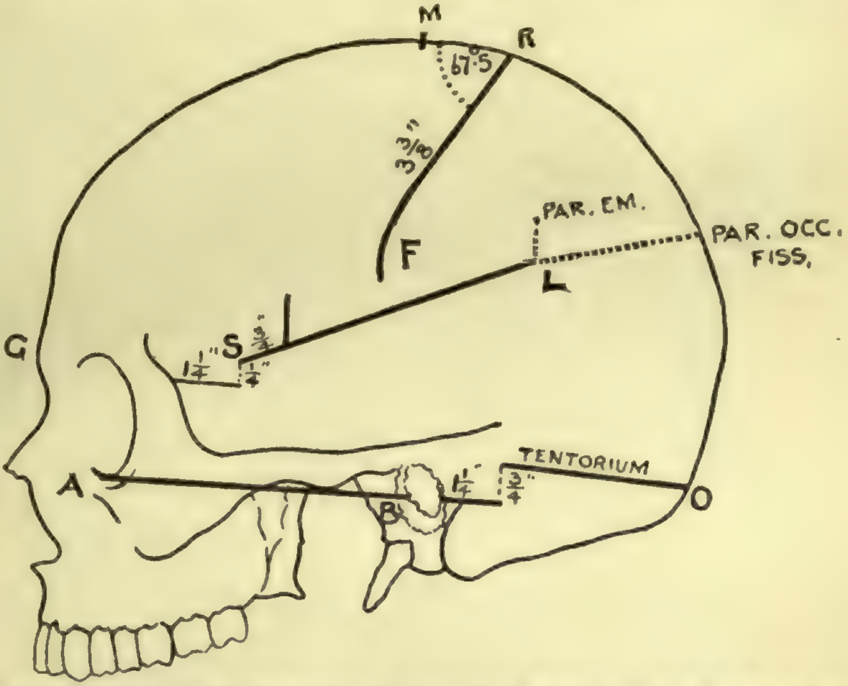

FIG. 1.—Diagram to indicate methods for finding the position of the fissure of Rolando (FR), the fissure of Sylvius (SL), the parieto-occipital fissure, and the line of attachment of the tentorium cerebelli to the inside of the skull.

The following are the areas of the brain with which the surgeon is chiefly concerned:—

(1) The sensori-motor or Rolandic area; (2) the areas for speech; (3) the area for sight; (4) the area for hearing; (5) the area for smell and taste. The position and functions of these various centres have already been indicated (p. 12).

CEREBRO-CRANIAL TOPOGRAPHY.—It is necessary to ascertain the relation of the various centres on the cortex to definite points on the surface of the skull, so that the surgeon may be able to accurately expose any such centre with the trephine.

As those parts of the cortex, of which the functions are hitherto best known, lie for the most part in close relation to two important fissures, viz. those of Rolando and Sylvius, it will be sufficient for our purpose if we indicate a simple plan for locating these fissures, and one or two other points of the brain, in their relation to the skull surface.

Fissure of Rolando.—Lay down a line from the glabella (G) to the external occipital protuberance (O). Bisect this line at M, and take a point (R) half an inch behind its middle point. This corresponds to the upper end of the fissure of Rolando.

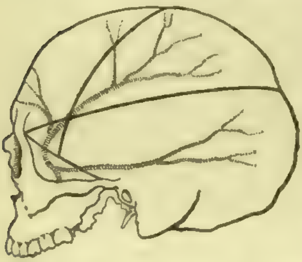

FIG. 2.—Diagram to show Chiene's method for determining the position of the anterior branch of the middle meningeal artery.

Draw a line 3⅜ inches long, downwards and forwards, at an angle of 67·5° with the line GO to the point F. The line FR will coincide almost exactly with the fissure of Rolando; the fissure, however, in its lower third turns a little backward and becomes slightly more vertical than a straight line between R and F.

The angle of 67·5° can easily be obtained by folding a square sheet of paper upon itself so as to make a triangle; this gives you the angle of 45° at the corner by halving the right angle of 90°. By folding the paper again upon itself in the same direction so as to halve the angle of 45°, you now obtain the right angle of the square subdivided into four angles of 22·5° each. Three of these angles taken together make up the required angle of 67·5°, and by applying the paper to the skull in the requisite manner, you can, with sufficient accuracy, determine the line of the Rolandic fissure (Chiene). Horsley's metal cyrtometer, composed of two strips of flexible metal fixed to one another at an angle of 67°, may be used for the same purpose, but it is unnecessary.

The Fissure of Sylvius.—Draw a base line (AB) from the lower margin of the orbit back to the auditory meatus.

Then draw a second line parallel to and above AB, from the external angular process of the frontal bone backwards 1¼ inches. Take a point S ¼ inch above the posterior extremity of this line, and this will correspond to the anterior extremity of the fissure of Sylvius.

Next draw a line from S to a point L, ¾ inch directly below the summit of the parietal eminence; the line SL will lie over the fissure of Sylvius; the short anterior limb of the fissure runs upwards, almost vertically, for about 1 inch, from a point ¾ inch from the anterior extremity of the line SL. The fissure of Sylvius is about 4 inches long.

If the line SL be produced backwards to the middle line of the head so as to meet the line GO, the point of their junction will indicate the position of the *parieto-occipital* fissure. This is useful in determining the anterior margin of the occipital lobe in cases of interference with vision due to brain lesion.

The tentorium cerebelli is attached to the skull along a line drawn from a point 1¼ inch behind and ¾ inch above the external auditory meatus to a point ½ inch above the external occipital protuberance.

The left hemisphere, being in right-handed persons more developed than the right hemisphere, is often found to extend beyond the middle line of the head as drawn from the glabella to the occipital protuberance.

To find the *anterior branch of the middle meningeal artery* (a vessel from which intracranial hæmorrhage is very common) draw a *first* line from the external angular process of the frontal to the parieto-occipital fissure. Draw a *second* line from the external angular process to the root of the zygoma immediately above and in front of the external auditory meatus. Bisect this line, and from its centre draw a *third* line to the midpoint between the glabella and inion. At the point where this *third* line intersects the *first* line will be found the vessel in question (Chiene).

OPERATIVE MEASURES.—While the description of some of the details of operative procedure in the case of the various conditions of disease and injury of the brain may be found under the different headings of epilepsy, cerebral tumour, etc., it will be as well at this stage to lay down rules which will apply generally to surgical operations on the brain.

Preparation of the Patient.—The patient should be kept in bed for a few days before operation; a low, digestible, diet given; the bowels should be opened on the morning of the operation by a purgative given the night before, and, if necessary, by an enema in the morning.

If he has been taking bromide of potassium for epilepsy, it is as well to stop it several days before operation, as it lessens his power of resisting the shock, which is often very severe.

The head should be completely shaved at least twenty-four hours before operation, repeatedly washed with soap and water, then scrubbed with turpentine, and subsequently with sulphuric ether. A compress of sterilised gauze wrung out of a solution of formalin, corrosive sublimate (1 in 2000) or carbolic acid (1 in 40), covered with mackintosh, should then be applied at least twelve hours before operation, and must be kept on until the commencement of the operation.

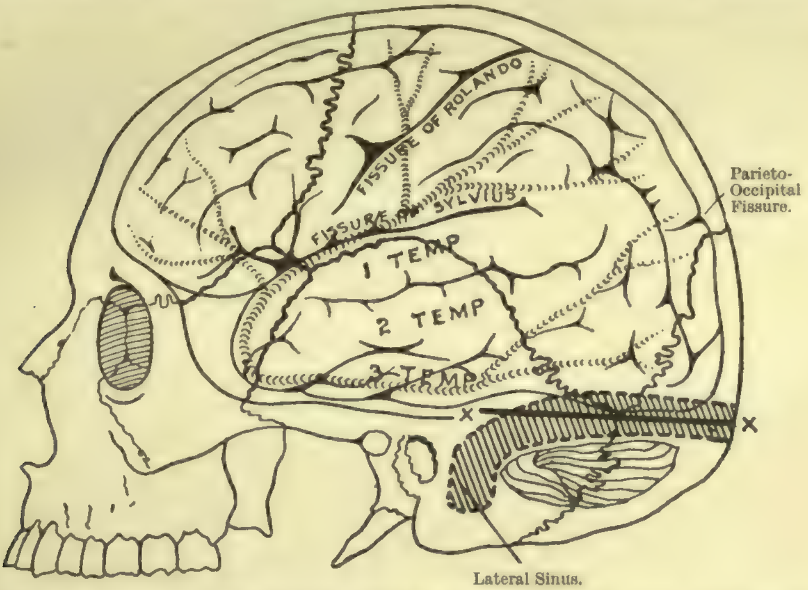

FIG. 3.—Diagram showing relation of brain to the fissures of the skull, and to the middle meningeal artery. x-x, line of attachment of tentorium to inner aspect of the skull.

Before this compress is put on, it is as well to measure and mark out on the scalp with nitrate of silver, or a sterilised aniline pencil, any line required, such as the fissure of Rolando or fissure of Sylvius.

Immediately before the operation a hypodermic injection of from $\frac{1}{6}$ to $\frac{1}{3}$ grain of morphia should be administered; this has the double object of lessening hæmorrhage from the brain by contracting the arterioles, and of allowing the patient to be kept unconscious during the operation with a much smaller quantity of the anæsthetic.

There is no question that, where an *anæsthetic* is necessary, as it always is unless the patient be comatose, chloroform is the best, as its use is accompanied by much less congestion of the cerebral vessels than when ether is given.

DETAILS OF OPERATION.—The head should be kept fairly high, and on a good firm pillow, so as to lessen hæmorrhage.

When the patient is under the influence of the anæsthetic, the surgeon, having taken scrupulous care to render his hands, instruments, etc., aseptic, first bores a hole with an awl, or other suitable instrument, through the scalp into the skull at one or more points, so as to accurately mark out on the skull the exact line of the fissure of Rolando, or any other region he may wish to identify, corresponding with the marks he has already made on the surface of the scalp. When the scalp flap is retracted, he will then have these marks on the bone to guide him to the area which he wishes to expose.

The Incision in the Scalp.—The scalp being then once more carefully washed with lysol solution (1 in 100) or carbolic solution (1 in 40), the surgeon makes a horse-shoe flap over the site which he wishes to trephine.[1] The flap should be a good deal bigger than the expected trephine opening, and its base should be so arranged as to include, as far as possible, the chief arteries and nerves coming to the part, so that the blood and nerve supply shall be interfered with as little as may be. As a rule, it is as well to leave the pericranium on the bone and not attempt to remove it with the flap of scalp; but this does not matter much. The usual plan is to open the pericranium by a separate crucial incision sufficiently to allow the trephine to be applied to the uncovered bone.

The Opening of the Bone.—In trephining the skull the following points are worth remembering:—

Do not make too small an opening. For the purpose of exploring the brain or meninges a trephine of at least 1 to 1½ inches in diameter is advisable.

As asepsis is of great importance in brain surgery, and as the old-fashioned trephines are very difficult to sterilise, it is safer to use an instrument, such as Galt's or Horsley's trephine, which can be taken easily to pieces, and which can be rendered safe by boiling.

One must bear in mind that different skulls vary enormously in thickness, and that the individual normal skull varies very much in this respect at different parts. Thus the frontal bone is throughout usually very much thicker than the inferior part of the parietal, or that part of the occipital on each side of the middle line. Again, the diploe is absent in most of the squamous bone and in the occipital fossæ, and is scarcely to be recognised in the case of very old or very young patients.

In applying the trephine one should avoid, if possible, placing it over the sinuses or large branches of the meningeal artery.

Taking into consideration the convexity of the skull and its very varying thickness at different points, it is necessary to be careful in using the trephine to make it bear equally all round the cutting surface, and to test the depth of the groove every now and then with a probe or quill so as to make sure that the dura mater or brain be not damaged.

If the probe indicates that one part of the groove is deeper than the rest, or that the bone is cut completely through, care must be taken that the trephine be slanted so as not to bear on that part of the groove.

It is important not to use any force in attempting to lift out the disc of bone with the elevator before it is completely, or at least very nearly completely, cut through. If this mistake be made the disc is apt to split in half through the diploe, the inner table being left behind at the bottom of the wound, and giving considerable trouble to remove.

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[1] The old crucial incision is now abandoned as less convenient and more apt to be followed by hernia cerebri. Where there is already a wound, as in some cases of compound fracture, this wound may be enlarged if necessary in any direction; but where possible, it is as well not to have the stitches in the scalp directly over the gap in the bone, but to adhere to the principle of the horse-shoe flap as far as may be.

Smart bleeding occasionally occurs during the operation of trephining from the vessels in the diploe. If at all severe, it may be stopped either by crushing the bone opposite the bleeding spot with a strong pair of forceps, by plugging the bleeding vessel with a small piece of sterilised gauze or sponge, or, perhaps best, by Horsley's putty, which is composed of carbolic acid, 1 part; oil, 2 parts; and wax, 7 parts. This material, or sterilised wax, should always be at hand when the skull is to be opened.

When, for any reason, the original trephine opening is not found sufficient for the purpose in hand, it may be enlarged by Hoffmann's gouge-forceps, the bone being thus quickly nibbled away over a sufficient area; or a second trephine opening may be made at a short distance from the first one, and the bridge of bone between them cut across with Hey's saw, and removed with powerful bone pliers.

Such a bridge of bone may also be quickly cut through by a rotatory saw attached to a dental engine or electric motor, or by the spiral osteotome of Dr Cryer. Gigli's wire saw, with which the bone is cut from within outward, the instrument being passed in at one trephine opening and out at a second, may also be used. In using these instruments special precautions must be taken to prevent injury to the dura mater.

The chisel and mallet, or gouge, are favourite instruments for opening up the skull with some surgeons. The series of blows inflicted by the mallet would appear to be a very serious and unnecessary addition to the shock often inseparable from the operation of trephining; still the method finds favour with many American and German surgeons.

If the gouge be used, extreme care must be taken that it does not plunge through the inner table and wound the subjacent parts.

When it has been found necessary to remove with the trephine a large piece of the skull, and the brain is thus uncovered by bone over a considerable area, not only is the patient liable to suffer severely, or even fatally, from blows on the unprotected brain, but a certain number of such patients experience troublesome vertigo on stooping.

For these reasons it has been recommended to put back the pieces of skull removed by the trephine in the hope that they may survive, and by adhering to surrounding parts may restore the normal protective covering to the brain.

If this plan be adopted, the pieces of bone should on removal be at once put into a warm aseptic, or 1 per cent sterilised saline solution, and kept at a temperature of about 100° till they are replaced in the wound at the end of the operation. Some surgeons put back the discs of bones as they are, while others recommend cutting them up into small fragments and planting these over the surface of the dura before stitching up the wound.

There are several objections to either plan, viz. the fragments of bone may either become absorbed, or may necrose, and give trouble in that way, while the bony covering they form is, at the best, of little protective value. When the disc is put back as a whole there is a serious risk of its becoming depressed below the level of the surrounding skull and thus pressing upon the brain. The writer has operated on two cases where epilepsy was caused by such depressed fragments of skull, the result of previous operations.

Where there is only a single disc of bone removed, and that of a diameter not exceeding $1\frac{1}{2}$ inches or so, there is no necessity to take any precautions, as there is always a certain amount of bony outgrowth from the cut surface of the skull, which will partly close such an opening, and the risk of injury to the small exposed area left is trifling.

Where a large surface is exposed, it is better to make the patient wear a celluloid or light metal plate in the cap, covering the hole in the skull, so as to avoid injury. By far the best plan, however, is to operate by the osteoplastic method devised by Wagner, in which a flap of bone is lifted up without being separated from its attachment to the scalp, and in this way its vitality is insured when it is replaced.

Wagner's original method was to make a horse-shoe incision down to the bone over the part of the bin to be expraosed. Then, without lifting the scalp flap from the skull, he applied the gouge to the bone at the bottom of this incision all round the horse-shoe. He then insinuated elevators under the bone and cracked the base of the bony flap through, thus lifting up a window composed of both skull and scalp adhering to one another. The brain is thus exposed for the purposes of exploration or surgical interference, and the flap being replaced at the end of the operation, adheres firmly, and the gap is most thoroughly and satisfactorily filled up.

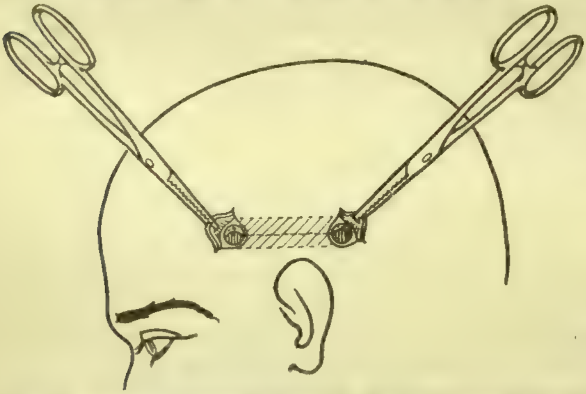

Fig. A.—Method of operating by bone flap. The first incisions at base of Ω. The shaded portion between the two trephine openings indicates the "tunnel" along which the saw is applied to the neck of the bone flap.

The writer has found by experiment on the dead body that a great deal of force is required to crack through the base of the bony flap (unless it is very narrow), and that splitting of the skull downwards towards the base may take place from such force being applied. In one case the split ran through the foramen spinosum, and would in the living body have given rise to troublesome bleeding. He has, therefore, devised the following modification of Wagner's operation, which is found to be satisfactory :—

The size and position of the flap to be raised being determined, an Ω-shaped incision is so planned that through its base may come the best possible blood-supply to the flap.

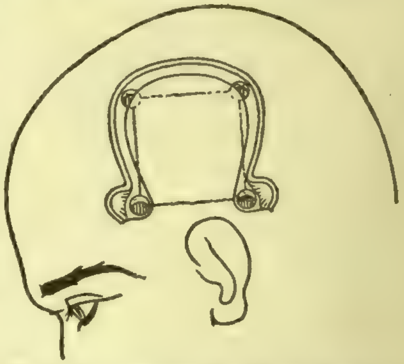

Fig. B.—The completed skin incision, with the four trephine openings in the skull, joined by saw-cuts.

Begin the Ω by making two small V-shaped incisions at each side of its base. Hold back with forceps these small flaps, and apply a ¾-inch trephine to each exposed area of bone, and take out the disc. Pass a director from one trephine opening to the other, *between the pericranium and the skull*, and lift up the soft parts off the bone sufficiently to make a small tunnel, along which a saw (shaped like a laminectomy saw, but finer) can be passed, with which the *neck* of the bony flap can be sawn through; the dura should, meanwhile, be protected from the saw by a second director passed from one trephine hole to the other *between the skull and the dura mater.*

The V-shaped incisions are then joined by the curved part of the incision, so as to complete the Ω; bleeding may be conveniently stopped from the flap itself by applying a clamp (such as Makin's intestinal clamp) across the neck of the flap, along the "tunnel."

The small trephine may now again be applied at the two upper corners of the Ω. A director is passed between the skull and the dura so as to protect the latter, and with a Hey's saw, an electric motor saw, Gigli's wire saw, or with a rotatory saw attached to a dental engine, the bone is cut through from one trephine opening to the other along the line of the incision in the soft parts. The bony flap attached to the scalp may then be gently lifted up without any force, and the dura exposed.

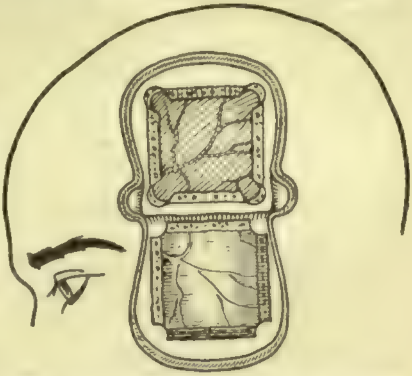

Fig. C.—The flap of bone and adherent soft parts turned down.

It is very important, in sawing through the bone, to *apply the saw obliquely* in such a way that the flap of bone to be lifted up shall be *bevelled at the expense of its under surface.* In this way, when it is replaced, it is quite certain that it cannot become depressed below the surrounding bone and cause irritation, while the stitches in the scalp hold the bone down in accurate apposition. Bony union takes place with certainty and rapidity.

Of course, in some cases, such as compound comminuted fracture of the skull, the method is not applicable; and where the operation is immediately followed by herniation of the brain from unrelieved intracranial pressure (such as is found, for example, in cases of basal tumours which cannot be removed), it may be found impossible to fit down the bony flap into its place again at the end of the operation. In such a case the piece of bone must simply be torn off the flap, and the scalp tissues stitched over the protrusion.

The *dura mater may be divided* either by a crucial or by a horse-shoe incision. In either case large vessels should be tied before they are divided, by passing beneath them very fine Chinese silk or catgut threaded on a curved round needle (such as is used for stitching intestine).

In making the horse-shoe incision through the dura, it is as well to let it open the opposite way to the flap in the bone, so as, when replaced, to increase its efficiency in preventing hernia. The incision in the dura should be not less than half an inch from the edge of the bone, so as to enable the surgeon to stitch it up easily.

To open the dura it should be first carefully punctured at one point with a bistoury, and then a small pair of curved blunt-pointed scissors used for the rest of the incision. The dura should be carefully lifted from the brain, as it may be adherent to it, and any violence used might cause bleeding from the large veins in the pia mater.

The tension of the dura mater should be noted, any marked convexity of its surface at the bottom of the trephine opening indicating abnormal intracranial pressure, and calling for special care while incising the dura not to wound the subjacent brain or vessels.

The surface of the brain which is now exposed should be examined—

(*a*) By *inspection.*—The presence or absence of pulsation (the latter denoting increased intracranial pressure) should be noted.

A yellow or livid appearance of the cortex may denote a subjacent growth (Horsley), while evidences of chronic or recent irritation, œdema, new growths, etc., may be visible.

(*b*) By *palpation*—When the increased resistance of a hard tumour, or fluctuation in a cyst or abscess may often be made out.

(*c*) By *exploration* with a fine hollow exploring needle.—This may be safely thrust into the substance of the brain in several directions, but care must be taken not to move the point of the needle about laterally in the brain substance, or else of course it will cause considerable laceration. The needle must be inserted by a direct stab, and be drawn out in the same line.

By this means the presence of a cyst or abscess may be ascertained, in which case a pair of sinus forceps should be slipped along the exploring needle while it is still in position, an opening thus made, and a drainage tube inserted.

(*d*) By *incision.*—Where there are suspicions of a growth underlying the cortex, this may be divided for the purpose of further exploration.

The incision into the cortex should be made with a blunt instrument, such as a periosteum elevator, and the best situation for such an incision is at the summit of a convolution. The cut should go directly in at right angles to the brain surface so as to damage vessels as little as possible.

In the case of *abscess in the brain*, a large india-rubber drainage tube should be inserted and brought out through the lower angle in the scalp flap. This should be left in probably for several days or even weeks, and should not be removed until there are evidences of the abscess cavity being obliterated. It is most important to remember not to syringe out such a cavity with any force, or laceration of the brain will happen.

In the case of *cyst of the brain*, if near the surface it may be dissected out, or if extensive, or deeply situated, may be opened and drained, or else plugged and allowed to heal by granulation.

In the case of *tumour of the brain*, if on or near the surface, and encapsulated, the growth can be easily shelled out with the finger; or if it be of the infiltrating variety, such as a glioma, the tumour must be excised with a blunt knife of some kind, a small margin (say ¼ inch) of apparently healthy brain tissue all round the growth being cut away with the tumour.

The cavity left by the removal of a tumour fills up considerably at once by the bulging into it of the subjacent brain tissue. If, in spite of this, a considerable cavity still be left, it may either be allowed to fill with blood-clot, which may become organised later, or it may be plugged with gauze. If the bleeding is at all smart, or asepsis doubtful, the latter plan had better be adopted.

Hæmorrhage from the vessels in the pia mater after removal of brain tumours is occasionally severe. If pressure, plugging with gauze, hot lotion, and the like, be not effectual, attempts may be made to ligature the vessel with fine catgut. In the case of bleeding from the brain substance itself pressure with a plug usually succeeds, though in rare cases styptic applications or a touch with the cautery may be necessary. In the case of wound of a large sinus, pressure forceps are sometimes left on the vessel for some days when other means have failed.

The brain should be handled as little and as gently as possible, so as to avoid shock. Horsley advocates the principle of doing these operations *in two stages*, the dura being exposed at the first sitting, and the tumour removed at the second, so as far as possible to minimise shock. There should be no hard and fast rule on this point; the surgeon should judge whether or not it is well to delay removal of the growth till a second operation, by taking into consideration the amount of blood lost in the preliminary stage, the general condition of the patient, and so forth. After operations on the brain, *drainage*, as a rule, is not called for. If, as stated above, there be a large cavity a drain may be left in for a few days, and in cases of abscess it is always necessary, otherwise it is safer to close the wound entirely.

The surface of the brain being thoroughly douched with warm boric or sterilised saline solution, the dura should be stitched in position with very fine Chinese silk, and the superficial flap adjusted and kept in position by horse-hair, silver wire, or other sutures.

INJURIES TO THE BRAIN

The comparative immunity from injury enjoyed by the brain and spinal cord is due in part to their effective bony and membranous coverings, but also in great measure to the elastic cushion or water bed provided for their protection by the cerebro-spinal fluid.

This fluid has another very important function, namely, the regulation of the amount of intracranial pressure on the brain.

It is known that the cerebro-spinal fluid permeates the whole substance of the brain and spinal cord, entering by the lymphatic sheaths which surround the cerebral vessels, and finding its way along these vessels to certain lymphatic spaces surrounding the nerve cells; there is also a free communication through the foramina of Majendie and those in the roof of the fourth ventricle, between the fluid in the subarachnoid space and that in the ventricles.

Further, it has been discovered that the cerebro-spinal fluid is capable of leaving the cranial cavity and passing into the spinal canal, the theca vertebralis being capable of considerable distension for its accommodation; and, again, a certain amount may escape from the cerebro-spinal cavity by passing along certain lymph tracks, which, for the most part, are in connection with the cerebro-spinal nerve sheaths as they leave the brain or spinal cord. Some twenty-one cases have also been recorded by St. Clair Thompson in which there was a spontaneous escape of cerebro-spinal fluid from the nose. The writer has had one such case under observation, and in this, as in most of the recorded cases, the exudation of cerebro-spinal fluid seemed to have a marked relation to an existing increase of intracranial pressure.

A consideration of the intimate relation of the cerebro-spinal fluid to all parts of the brain and cord, and of the power it possesses of shifting its position according to circumstances, suggests very forcibly its value as a regulator of intracranial pressure. It is evident that it is a factor to be borne carefully in mind in the consideration of injury or disease of the brain.

Injuries of the brain have a special interest of their own, in so far that, while of course they may be of all grades of severity, from slight concussion to such as are immediately fatal, their effects on this sensitive organ are frequently so far reaching, and so much out of proportion to their apparent severity, that they all demand the most careful investigation and treatment.

Injuries may be inflicted over a limited area of the brain, and the lesion may be such as to interfere exclusively with the functions of a distinct and definite centre. In these cases a consideration of the symptoms in the light of our knowledge of localisation of function in the brain may lead us to a diagnosis of the exact position of the lesion.

Generally speaking, a comparatively slight and local irritation of a motor centre will cause spasm of the muscles represented at that centre, while a more complete destruction of the centre will lead to paralysis of the muscles concerned. It is necessary to remember, however, that even if a motor centre be entirely removed by injury or operation, other parts of the brain may subsequently take up the work of the damaged centre to a very considerable extent.

As the result of injury hæmorrhages may take place upon the surface or into the substance of the brain, and these in their turn may cause compression of the brain or lead to the formation of cysts.

One of the most frequent general conditions following injury to the brain is that of concussion, while contusion or laceration of its substance are followed by a definite train of symptoms.

Inflammation of the brain and its membranes, followed it may be by softening, sclerosis, or abscess formation, are occasional results of severe injury.

Concussion of the Brain

This term signifies a state of more or less complete arrest of the functions of the brain following injury to the head, and characterised by many of the symptoms usually associated with the condition of shock or collapse.

It is usually caused by a severe blow or fall on the head, but may follow a fall on the buttocks or even on the feet.

The *pathology* of this condition, which is somewhat obscure, may in part at least be gathered from the chief post-mortem appearances, which are as follows:—

(*a*) Minute punctiform hæmorrhages scattered throughout the brain, and in some cases more distinct extravasations of blood, or areas of actual disorganisation of brain tissue.

(*b*) Anæmia of the brain. It has been suggested by Duret that this is due to the force applied to the skull causing a temporary depression on the surface of the bone, by which means the subjacent brain is compressed, and the cerebro-spinal fluid is forced out of the lateral ventricles and lymph spaces towards the base of the skull, and particularly into the fourth ventricle, the distension of which, by stimulating the restiform bodies, produces anæmia of the brain.

(*c*) Engorgement of the right side of the heart, lungs, and other internal organs. This seems to be due to vasomotor disturbances in the medullary centres, and consequent gravitation of blood to the portal system.

Symptoms.—These vary with the severity of the injury. In mild cases all that is noticed is a temporary or momentary unconsciousness or giddiness, with a little confusion of mind, and occasionally a certain degree of pallor. These symptoms may be succeeded by headache more or less persistent and severe.

In well-marked and more severe cases there is, immediately following the blow, sudden unconsciousness, more or less complete, and lasting, it may be, several minutes, hours, or even days.

During this time the patient may usually be roused by shouting in his ear, or by other strong stimuli, but in some cases the unconsciousness is complete. He usually lies flat on his back; his muscles are relaxed; the pulse is weak and fluttering; the respiration is irregular, shallow, and slow, occasionally marked by deep-drawn sighs; the temperature is subnormal.

The eyes are shut, and the conjunctival reflex is usually absent; the pupils are usually (but not always) contracted, and, except in very severe cases, they react to light.

Other reflexes are usually present, unless the concussion is very severe, but they are usually slow, and somewhat difficult to elicit.

The sphincters are often relaxed, and unconscious passage of urine and fæces may take place.

The foregoing symptoms constitute what is called the *first stage* of concussion, sometimes called the *stage of collapse.* It may last for a very variable time, and may then pass, on the one hand, into more profound unconsciousness, in which the patient gradually sinks and dies; or the pendulum may swing back from collapse towards the other extreme, namely, irritation or inflammation of the brain, followed it may be by symptoms of compression.

This state of things may be looked upon as an exaggeration of what takes place in all favourable cases during their *second stage*, or *stage of reaction.*

This stage is characterised, first and foremost, by sudden vomiting; in rarer cases by an epileptiform seizure. The patient, who usually feels chilly when he first begins to regain consciousness, soon becomes warmer and of a better colour, his pulse and respiration improve, and consciousness returns.

The temperature is now apt to swing beyond the normal to about 100°, and the pulse to become full and bounding; headache is likely to supervene, and may last for some days. This state of matters, lasting for a variable time, tends in favourable cases to gradually subside to the normal condition, but on the other hand may go on to encephalitis, meningitis, or to more or less permanent interference with some brain function, such as loss of memory, incapacity for work, or to neurasthenia.

Contusion or laceration of the brain may be the result of a localised injury caused by the direct blow of a hammer, bullet, or some such instrument, smashing through the skull and impinging directly on the brain; or may be due to violence applied to the outside of the cranium, causing the brain to impinge against the inner surface of that side of the skull opposite to the site of injury. When this happens the brain is said to suffer by "*contre-coup*," and the injury affects a considerable area of brain.

When the injury is strictly localised its effects may be also purely local, such as interference with some motor or sensory centre of the brain.

But in the large majority of cases an injury which is severe enough to contuse or lacerate the brain will also suffice to produce the condition of concussion, while the laceration of vessels frequently gives rise to hæmorrhage followed by symptoms of compression. One usually therefore finds, at any rate for the first few days, the symptoms of laceration or contusion accompanied by those of concussion and even of compression.

When, however, the patient lives long enough, and the symptoms of compression do not mask entirely those of laceration, it usually happens from the second to the fifth or sixth day that characteristic symptoms of laceration show themselves in the shape of spasm or rigidity in certain groups of muscles. This, of course, will only be the case where the site of injury is in the sensori-motor area, and appears to be due to the supervention of inflammation or spreading œdema in the injured part. Where the laceration is very severe, and disorganisation of the centre has taken place, paralysis may take the place of spasm.

Laceration of the brain may be followed by yellow softening, œdema, or abscess formation, while cysts occasionally form as the result of hæmorrhages on the surface or in the substances of the brain. These conditions, again, may be followed by traumatic epilepsy, motor or sensory paralyses, etc.

One curious and not infrequent result of laceration of the brain is the condition known as *cerebral irritation.* It may come on a few hours, or more commonly two or three days after the accident, and is supposed to be most frequently met with in injury to the frontal lobes, which are the seat of the intellectual and mental functions. The patient lies on his side, and will not be persuaded to lie on his back. His knees are drawn up and his arms flexed. The eyes are tightly shut, and he resents extremely any attempt on the part of the surgeon to open them for purposes of examination.

The pupils are contracted and react to light; the pulse is weak and somewhat slow; the temperature not far from normal; the urine and fæces may be passed in bed.

The patient tosses about in bed in a restless way, takes no interest whatever in what is going on about him, and can hardly be got to answer any question. He is in a condition of extreme irritability, and if disturbed he frequently swears in the most profuse manner.

This condition may pass off in a few days, the patient becoming less irritable, and the other symptoms gradually disappearing. In other cases serious and permanent damage results to his intellectual faculties, while in some instances meningitis may ensue.

The condition known as *spreading œdema* of the brain occasionally follows injury to the brain, such as contusion or laceration. As a result of effusion of blood or serum, or the pressure of depressed bone, or abscess (or even a tumour), the circulation in the veins of the primarily affected part is hampered; this leads to further local effusion of serum, followed in turn by more pressure, and so on. In this way a large part of the brain may become progressively œdematous, the process spreading from an original point of pressure or irritation. The chief clinical importance of spreading œdema is that it gives rise to symptoms of compression of the brain.

In severe cases of concussion or contusion of the brain, it is frequently noted that the recollection not only of the accident itself, but also of what may have happened some minutes, hours, or even days before the accident, is entirely blotted out of the patient's memory. In most cases this is regained, but in some the loss is permanent.

Treatment of Concussion.—In the *first stage* the symptoms of shock must be met by keeping the head low, and applying warmth and friction to the surface of the body; a warm water enema may be of some use, but alcoholic stimulants are only to be given with great caution in cases of profound and obstinate collapse, as their use may induce too severe reaction, and tend to produce hæmorrhage or inflammatory action. A passing stimulant, such as aromatic spirit of ammonia, or inhalation of strong smelling salts, is less objectionable.

Such drugs as strychnia and strophanthus used hypodermically must be reserved for desperate cases. Nothing must be given by the mouth until the patient is sufficiently conscious to swallow. Irrigation of the head with hot water is said by Horsley to be of great use in bringing about reaction in cases of severe concussion.

The treatment of the *second stage* is to be directed towards preventing excessive reaction. The patient should be kept in bed in a dark and quiet room, his diet should be light, his bowels should be kept freely open, and on no account should he be allowed to read or transact any business until convalescence is well established. The danger of allowing a patient to use his brain during the stage of reaction after a severe concussion can hardly be exaggerated. Brain abscess, neurasthenia, or even insanity are some of the probable results of this indiscretion. If the reaction be excessive and meningitis seem to be impending, leeches or blisters should be applied behind the ears, and a large dose of calomel given.

The *treatment of contusion and laceration* of the brain is practically identical with that of concussion. If symptoms of inflammation, abscess, or compression arise they must be dealt with in the way described under those headings.

In cases of *cerebral irritation* the use of an ice-bag to the shaved head, small doses of bromide of ammonium, and a digestible but nutritious diet, are the chief additional points of importance.

Compression of the Brain

Compression of the brain may be either general or local.

"General compression," or, as it is more commonly called, "increased intracranial pressure," signifies increased pressure of the cerebro-spinal fluid on the whole central nervous system. This condition is met with in such cases as hydrocephalus, meningitis with effusion under the arachnoid, or tumours situated at the base of the brain, which by their pressure lead to distension of the ventricles and subarachnoidal space with an excess of cerebro-spinal fluid. In other cases general compression may occur as a complication or sequela of local compression.

By the term "local compression" is meant a direct localised pressure on some part of the brain itself, frequently the result of injury, and characterised by symptoms usually associated with coma.

The term "cerebral compression" should be restricted to this latter variety.

Increased intracranial and intraspinal pressure due to such causes as hydrocephalus, meningitis, or tumours, will be discussed under these headings. Here we are chiefly concerned with the common form of compression due to injury.

The usual *causes* of compression after injury are:—

1. Inflammation of the brain or membranes, frequently the result of septic infection.
2. Intracranial hæmorrhage.
3. Depressed portions of the skull (rare).

The *symptoms* of compression vary very much with different degrees of pressure, and according to the exact locality in the brain to which pressure is applied. Thus we find all degrees, from slight headache or drowsiness to profound coma, resulting from different intensity of pressure; while, as regards locality of lesion, pressure on the frontal lobes may give little sign, while pressure on the motor area of the brain will cause paralysis in the corresponding group of muscles.

The *temperature* is usually subnormal, often reaching as low as 95° F. to 96° F. This is a most reliable and valuable sign of general compression of the brain, but it must be borne in mind that in the later stages of a bad case the temperature may become very high, and death may occur with the temperature at 106° F., or even higher.

If the compression be general the temperature on both sides of the body is equal; if the pressure affect one hemisphere of the brain only, there will usually be a rise of temperature of about one degree Fahrenheit on the side of the body opposite to the compressed hemisphere.

Here, again, in very bad cases the rule may be reversed, and the temperature be lowered, instead of raised, upon the side of the body opposite the lesion.

The *pulse* is slow, full, and deliberate in an ordinary case. After a while, from exhaustion of inhibitory centres, it may become fast, weak, and irregular. This is a bad sign.

The *respiration* is, in moderate cases, slow, deep, and regular. In severer cases it becomes irregular, stertorous (owing to paralysis of the soft palate), and accompanied by puffing out of the cheeks and lips (due to paralysis of the facial muscles).

In still worse cases the respirations become of the Cheyne-Stokes' type.

Stoppage of the respiration is the immediate cause of death in cases of compression.

Nervous Symptoms.—In many cases at first the patient complains simply of headache, and this is due, apparently, to stretching of the dura mater. This headache tends to get worse, and to be succeeded by drowsiness. This important symptom, in its turn, becomes more pronounced, and eventually complete coma may gradually supervene.

Neuralgia, for example, of the branches of the fifth nerve, may result from pressure, while paralysis of the muscles supplied by the sixth, seventh, or other cranial nerves may occur from the same cause.

Motor paralysis may occur, either affecting a single group of muscles in cases of strictly localised brain pressure on the motor area, or as a hemiplegia, beginning on that side of the body opposite to the brain lesion, and tending to become general as the pressure spreads.

Convulsions may happen in groups of muscles if the corresponding centres in the brain be very suddenly compressed. Where the compression comes on gradually convulsions are not met with, but paralysis ensues. Even in those cases of sudden compression, where convulsions do occur, they soon give place to paralysis in the affected muscles.

The Eyes.—The pupils first contract, and then dilate and become insensitive to light. If the pressure on the two sides of the brain be equal, then both pupils contract and dilate at the same pace; if the pressure on the brain be unilateral, then the pupil of the eye on the same side as the lesion will go through the above changes of contraction and subsequent dilatation at a quicker rate than will the other pupil.

An ophthalmoscopic examination of the eye will often disclose choked disc, while from pressure on the cavernous sinus the veins at the back of the orbit may become so engorged as to cause a considerable amount of exophthalmos.

The *reflexes* are gradually lost as compression becomes worse. The bladder is paralysed, so there is retention of urine until the bladder gets quite full, when overflow takes place, and the water dribbles away.

The sphincter ani is paralysed, but as the rectum is not interfered with there may be unconscious evacuation of fæces.

Diagnosis.—If symptoms of compression come on immediately on receipt of an injury, they are probably due to a depressed fracture pressing directly on the brain. This condition is quite exceptional.

If the symptoms come on during the first few hours after injury, the most probable cause is hæmorrhage, usually from a branch of the middle meningeal artery.

If the symptoms are delayed in their appearance for several days, the compression is probably due to inflammatory exudation or spreading œdema.

If no symptoms appear for weeks or months, the most probable cause is chronic abscess of the brain.

In the case of cerebral tumours the brain has time to accommodate itself to the abnormal pressure by the safety-valve action of the cerebro-spinal fluid; we do not, accordingly, meet with typical symptoms of compression in tumour cases except in their latest stages, or where they are complicated by hæmorrhages.

In cases of contusion of the brain the common rule is monospasm followed by monoplegia; in compression the rule is paralysis unaccompanied by spasm, except in the comparatively rare cases where the compression comes on very suddenly.

Diagnosis from Concussion.—As concussion and compression are in many respects somewhat like each other, and as compression frequently calls for immediate surgical interference, it may be as well to indicate the chief diagnostic points of difference.

In concussion the symptoms come on suddenly, immediately after the injury, and suggest syncope.

In compression the symptoms come on gradually, some minutes or hours after injury, and suggest coma.

In concussion there is general relaxation of the muscles, which tends to pass off when reaction sets in.

In compression there is localised paralysis at first, tending to become general and absolute as the compression increases.

In concussion the respiration is shallow, slow, and sighing.

In compression it is stertorous, puffing, or of the Cheyne-Stokes' character.

In concussion the pupils are equal, and usually react to light.

In compression they are often unequal; they first contract and then dilate, and become insensitive to light.

In concussion there is incontinence of urine with an empty bladder.

In compression there is retention and overflow from a full bladder.

There are many other conditions of coma from which we must be able to distinguish that due to compression of the brain. The chief of these are—

1. *Cerebral Apoplexy.*—The exclusion of injury is here perhaps the chief point.

2. *Opium Poisoning.*—Here we find the pupils contracted to a pin-point until just before death, when they may dilate. (It must be remembered that a hæmorrhage into the pons is followed by pin-point pupils, high temperature, and sweating.) In the case of compression the pupils are usually unequal, at first contracted, then dilated and immobile. In opium poisoning the lividity of the face, and the smell and chemical examination of the contents of the stomach may give a clue.

3. *Alcohol Poisoning.*—A condition very like opium poisoning may result from a large dose of strong alcohol. Here we also find pin-point pupils, but if efforts are made to rouse the patient the pupils dilate, and then contract again as he again becomes unconscious. The absence of the typical stertor of compression, and a careful examination of the vomit and urine for alcohol, may give useful information; but in many of these cases there is great difficulty in diagnosis, as a patient who has taken a certain amount of alcohol may either receive an injury to the head causing compression, or may be seized with apoplexy; so it is a good rule in these cases to watch the patient carefully for twenty-four hours, or longer if necessary, when the diagnosis will usually be clear.

The diagnosis of other causes for coma simulating compression depends chiefly on a consideration of the history of the case. Thus in epilepsy, followed by coma, there may be a history of previous epileptic attacks; in uræmia the patient will probably have had convulsive seizures, while an examination of the urine may disclose some evidence of chronic renal disease; in meningitis the early stages of the disease are different from those of compression; in diabetic coma the pupils will react, and the breath and urine have a smell like chloroform, while the urine will be found abnormal.

Coma due to excessive heat or cold is usually sufficiently recognisable by the history of its causation.

Treatment of Compression.—This consists in the removal of the cause where that is feasible.

In the rare cases where a depressed fracture is causing the depression, trephining and elevation of the depressed fragment must be at once performed; and considering the frequency with which traumatic epilepsy follows, it may be after some lapse of time, upon depressed fracture, even where such depression is not enough to cause symptoms of compression, it is advisable to elevate portions of skull which are decidedly depressed, even though compression symptoms are not present at the moment. This rule applies in almost every case in the adult; possibly in the case of the more elastic and resilient skull of the child it is not so binding, as with them the depressed skull has a wonderful capacity for regaining its normal level without surgical intervention.

Abscesses of the brain must be opened and drained (see p. 94), and clots of blood causing compression must be removed in the manner described under "Intracranial Hæmorrhage."

In spreading œdema it has been suggested to open the dura and deeply incise the brain tissue, so as to afford relief of tension.

In case of tumour of the brain the tumour must be removed, or where that is impossible, the removal of a large area of the skull, with opening of the dura, and the encouragement of a hernia of the brain may be the only means to give relief.

Where, as in meningitis with effusion, the compression is general, and due to an increase in cerebro-spinal fluid, relief can often be afforded by draining the subarachnoid space or the lateral or fourth ventricles, or by drainage of the spinal canal by lumbar puncture or incision.

The other general indications for treatment are to keep the patient quiet in a dark and cool room, to open the bowels with croton oil or enema, to look to the condition of the bladder, and, if necessary, use the catheter, and to keep the patient sustained by nutritive enemata.

If cyanosis become very marked, free abstraction of blood by venesection is advisable.

Remember that death ultimately happens by failure of the respiration, and that it is possible by the use of artificial respiration to keep the patient alive for a considerable time, and so, possibly, to afford time for the performance of an operation for the relief of the compression.

INTRACRANIAL HÆMORRHAGE

Intracranial bleeding may be spontaneous or due to injury.

In spontaneous hæmorrhage or apoplexy the bleeding usually comes from the lenticulo-striate artery. Occasionally aneurysms of the cerebral arteries may give way and cause hæmorrhage, or vessels in close relation to tumours of the brain may be the source of the bleeding. In spontaneous hæmorrhage the bleeding usually takes place into the substance of the brain or into the ventricles, is usually out of reach of the surgeon, and, except by ligature of the common carotid artery on the same side as the lesion in cases which are becoming progressively worse, little can be done.

Traumatic intracranial hæmorrhage may occur either between the dura mater and the bone (subcranial), between the dura mater and the cortex (subdural), or into the substance of the brain or cavity of the ventricles (cerebral or intraventricular).

(1) *SUBCRANIAL OR EXTRADURAL HÆMORRHAGE.*—This is the commonest variety of intracranial hæmorrhage. In the large majority of cases the bleeding vessel is the middle meningeal artery. This vessel soon after its passage through the foramen spinosum divides into its anterior and posterior branches, and the bleeding point is usually either in the trunk of the vessel or in its anterior branch—the posterior branch being at fault only in about 7 per cent of the cases. The bleeding is usually caused by a fracture of the skull running through the anterior and lower part of the parietal bone, or by a spicule of inner table driven into the vessel; but occasionally bleeding from the middle meningeal artery or its branches may take place without any fracture, and may even happen on the side of the head opposite to the blow, in which case it is due to contre-coup.

Traumatic hæmorrhage may happen from wounds of the large sinuses of the brain, viz. the longitudinal, lateral, or cavernous, the last-named being probably the one most commonly damaged, as it lies in very close relation to the base of the skull, and is likely to be torn in basal fractures.

The veins of the diploe and middle meningeal veins are occasionally the source of hæmorrhage; and in very rare cases the internal carotid artery may be wounded by punctured wounds through the orbit, bullet wounds, or fractures of the base. When this happens ligature of the common carotid is the only resource.

Symptoms of Subcranial Hæmorrhage.—These depend very much on the size and nature of vessel wounded. While the force of arterial bleeding is sufficient to strip the dura mater from the skull, and so make room for a large clot which may cause symptoms of compression, the bleeding from a vein is more likely to be stopped by the resistance offered by the adhesion of the dura to the skull. Therefore large hæmorrhages when they are extradural are usually from the middle meningeal arteries, and not from the sinuses.

The injury which has caused the hæmorrhage may or may not be sufficient to stun the patient and cause temporary unconsciousness. If it do so the patient usually recovers his senses, and there is a "lucid interval" before the symptoms of compression commence.

This lucid interval is usually of some hours' duration, the time of the onset of compression symptoms depending on the size of the vessel wounded, and varying from an hour or two to two or three days.

The patient, who is usually stunned by the accident, recovers consciousness, but in an hour or two, as the clot enlarges and presses on the brain, becomes drowsy and stupid; this state of matters steadily progresses until complete coma is reached.

There is usually at first paralysis of that side of the body opposite the clot; and the pressure, which may at first be localised, for example, to the centres controlling the muscles of the face, may gradually extend up the Rolandic area affecting in turn the centres for the arm and leg, and eventually may cause a general compression of the whole hemisphere, and later of the whole brain, and a resulting paralysis of both sides of the body.

If the clot pass downwards, as it frequently does, towards the base of the brain, the pupil of the eye on the same side as the clot dilates and ceases to react to light.

Occasionally there may be protrusion of the eyeballs from pressure of the clot upon the cavernous sinuses.

It often happens that there are at first convulsive movements in the muscles corresponding to the brain centres pressed upon: these spasms, however, soon give way to paralysis.

If the effusion of blood be small in quantity it may be absorbed, though a certain amount of hemiplegia is usually left; if the effusion be a large one the compression is usually fatal unless relieved by operation.

Subdural Hæmorrhage. — Under this heading we may include hæmorrhages either between the dura and arachnoid, or between the arachnoid and pia mater. The symptoms and treatment are identical, and subdivision is therefore superfluous.

The most striking point of difference between the symptoms of extradural and intradural hæmorrhage is the fact that in the latter variety the onset of compression symptoms is very much more rapid, while the localising pressure symptoms are usually much less distinct.

The explanation of these facts lies in the circumstance that when bleeding takes place under the dura the arachnoid is usually wounded, and the blood pours out quickly into the subarachnoid space, there being now much less resistance to the outpouring of blood than in the case of extradural hæmorrhage where the adhesions of the dura to the skull offer considerable resistance. A large quantity of blood, then, being quickly poured out in a film of more or less equal thickness over a large part of the brain surface, there is not only a more rapid accession of compression symptoms, but these are apt to be more of the nature of general compression on a whole hemisphere than strictly localised to any particular region of the brain.

This same want of resistance to the bleeding in the case of subdural hæmorrhage allows the bleeding from a sinus, when it occurs in this position, to be as rapid and severe as when an artery is wounded.

Usually speaking, subdural or subarachnoid hæmorrhage comes from the rupture by contusion of small vessels in the pia, and especially from the branches of vein just as they are entering the longitudinal sinus; but it may also come from wounds of the middle cerebral or meningeal artery, or from the venous sinuses.

Where then the symptoms come on rapidly, and the lucid interval is absent, we should suspect a subdural hæmorrhage.

If the extravasation is not a large one recovery may take place; at the best it is slow, and apt to be incomplete. Cysts occasionally are formed in the clot, and are met with lying more or less loose in the subarachnoid space. In other cases irritation of the brain, epilepsy, or even insanity may supervene.

Cerebral Hæmorrhage.—Bleeding into the substance of the brain, or into the cavity of the ventricles, is rare as the result of injury. In the latter position it may come from severe concussion causing bleeding from the choroid plexus.

If the symptoms suggest intraventricular hæmorrhage a trocar might be inserted, and if blood were found, an effort might perhaps be made to remove the clot and stop the bleeding by plugging; or ligature of the carotid on the same side might be entertained.

Treatment of Intracranial Hæmorrhage.—In all cases of suspected intracranial hæmorrhage, where compression symptoms are becoming distinct, operate at once. This rule applies all the more forcibly if the hæmorrhage be subdural.

Operative Measures.—Make for the spot in the brain where the first symptoms of spasm, rigidity, or paralysis of muscles indicated that their corresponding centre was being pressed upon; and trephine here in preference to doing so at the site of apparent external injury, as the bleeding may be due to contrecoup. The localising symptoms are more reliable than the position of the external wound.

If the bleeding be from the middle meningeal artery it will probably be from the trunk or anterior branch. To expose these vessels, mark out a spot 1½ inches behind the external angular process of the frontal, and 1½ inches above the zygoma.

Shave and render the part aseptic; drill a hole through the scalp into the skull so as to mark the exact spot in the bone; turn down a flap containing the scalp and temporal muscle; incise the pericranium, and apply a trephine of at least one inch in diameter to the spot drilled in the anterior and lower corner of the parietal bone. Remove the disc, when the clot will usually bulge into the hole: scoop it away with some blunt instrument, and ligature the wounded artery by passing a suture ligature of fine silk or catgut beneath it. If it be impossible to tie the vessel as it lies in the bone, it may be plugged with sterilised wax or gauze. The middle meningeal artery may also be found by Chiene's method (p. 73). If the posterior branch of the middle meningeal be wounded, it may be reached by trephining at a spot immediately below the parietal eminence, and 1½ inches above the line of the zygoma produced backwards.

In most cases the dura should be opened and the brain examined, or a deeper hæmorrhage may be overlooked.

The clot will be usually found to be disc-shaped, of not more than three or four inches in diameter, thicker in the middle, and thinning off towards its edges. The subjacent brain is often considerably flattened or even hollowed out by pressure from the clot.

When the bleeding comes from a venous sinus it may usually be stopped by exposing the vessel with the trephine, and then plugging the sinus or applying a double ligature.

CEREBRAL TUMOURS

Chiefly owing to advancement of our knowledge in regard to the localisation of function in the brain, and to the increased safety of surgical procedure due to Lister, great strides have been made of late years in the diagnosis and treatment of cerebral tumours.

Still, it must be confessed that a great deal has yet to be done to clear up difficulties regarding the diagnosis, while the general consensus of opinion is that only from 5 to 10 per cent of cerebral tumours are amenable to surgical treatment.

Tumours of the brain may be true neoplasms, may be tuberculous or syphilitic deposits, or may be due to parasites.

Varieties of Cerebral Tumour.—*Fibromata.*—These are comparatively uncommon. They grow from the dura mater, are encapsulated and hard, slow in their growth, and their complete removal is followed by satisfactory results.

Psammomata.—These also are rare. They grow from the pia mater of the brain and cord, and are epithelial in character, surrounded by connective tissue, hard in consistence, of slow growth, and frequently calcified. They may develop in the choroid plexus, and are often found in the fourth ventricle, where they may give rise to serious pressure symptoms.

Sarcomata are very common, especially in adults. They grow from the dura mater or in the brain substance. They are usually of comparatively rapid growth; are encapsulated; somewhat firm in consistence; apt to be multiple, and may be secondary to sarcoma in other parts of the body.

Gliomata are also common, though somewhat rarer than sarcomata. They grow from the neuroglia of the brain; are usually single; are of rapid growth; of soft consistence (much the same as the brain tissue itself); are not encapsulated, but infiltrate the surrounding brain tissue; are very vascular; and the results of their removal is, as might be expected, unsatisfactory.

Glio-sarcomata.—Most gliomata contain numerous small round cells, and are in reality sarcomatous. Clinically glio-sarcomata may be considered practically identical with sarcomata.

Carcinomata usually occur secondarily to carcinoma in other parts of the body. They are soft, vascular, and unsatisfactory for surgical interference, not only because of the difficulty of their removal and malignancy, but on account of the original disease elsewhere. Primary carcinoma of the brain is rare.

Tuberculous deposits, either in the form of infiltrations or definite nodules, are very common, especially in children. They are most frequently found in the cerebellum or at the base of the brain; are usually multiple; are frequently accompanied by basal tuberculous meningitis; are usually of somewhat slow growth, but may become suddenly more urgent in their manifestations; are usually of firm consistence, but may soften at the centre. Occasionally they become quiescent, and may be partially absorbed, or shrivel up into a cicatricial mass, which by contraction and dragging on the brain substance may cause epileptiform attacks. They are notably incidental to childhood and young adult life, and frequently a tuberculous history is obtainable.

Syphilitic gummata are very common. They occur in adults; are usually cortical and on the upper half of the brain; they are often attached to the dura; other syphilitic manifestations are often evident, and a history of syphilis can probably be obtained; there is usually severe pain, especially at night, accompanied by loss of sleep; the use of iodide of potassium in large doses usually improves the symptoms in a marked way, and completes the diagnosis.

Parasitic tumours, usually in the form of cysts, occur more frequently in foreign countries than in Britain. They may be due to hydatids, echinococcus, and other parasites. They push the brain aside, and do not as a rule cause marked focal symptoms.

Other cystic tumours may be due to the degeneration of sarcomata or gliomata, or may result from localised hæmorrhages in connection with the brain or meninges.

In rare cases actinomycosis attacks the brain (see "Actinomycosis").

The *general* and *localising symptoms* of intracranial tumour are described on p. 49 *et seq.*

Treatment.—Before determining on operation it is necessary to diagnose as far as possible the situation and nature of the growth; to exclude cases which are inoperable because of the multiplicity of the growth, or because of similar growths in other parts of the body. It must be borne in mind that only some 7 per cent of all brain tumours are within reach of the surgeon, and that it is not possible to diagnose with certainty whether a case is cortical, subcortical, or both combined.

The ill-defined and infiltrating gliomata are most unsatisfactory; so much so, that some surgeons think it is not worth while removing them even when they are found. It would be of great value if one could diagnose a glioma from an encapsulated sarcoma, which latter is so much more favourable for operation. Allen Starr believes that the changes brought about by the application of cold, temporary hyperæmias, and suchlike causes, in the symptoms of a vascular growth like a glioma, may enable the surgeon to diagnose it from an encapsulated sarcoma.

Tuberculous tumours are very commonly multiple, and for this reason some surgeons are averse to operation. This is not, in most cases, a sufficient reason for refusing to operate. Intracranial pressure is relieved, and absorption of other tuberculous deposits may be stimulated by the operation.

In all cases where there is the smallest possibility of the tumour being syphilitic, large doses of iodide of potash (20 or 30 grains thrice daily) should be given for two or three months before deciding to operate. Mercury *may* also be exhibited. Two points must, however, be remembered: firstly, that while a gumma may be improved by antisyphilitic treatment up to a certain point, a cicatrix may be left which may cause epilepsy and demand treatment by operation; secondly, that, as we find elsewhere in the body, other tumours, malignant ones among the number, are often considerably improved for a time, and up to a certain point, by iodide of potash.

When once the surgeon has determined that the case is an operable one no unnecessary delay is permissible. Spreading œdema may occur at any time, and in other ways irreparable damage be done to the brain if the tumour be allowed to grow.

In cases which are doubtfully operable an exploratory operation may be justifiably recommended. Not only may the tumour when exposed prove more favourable than was indicated by the symptoms, but the removal of intracranial pressure is of the greatest value to the patient by relieving pain, retarding optic neuritis, prolonging life, and in some cases bringing about the absorption of tuberculous tumours. And it must be remembered that the medicinal treatment of cerebral tumours, except in the case of syphilis, is practically useless.

In cases of inoperable tumour, bromide of potassium, morphia, the application of ice to the head, and counter-irritants to the back of the neck, may be used as palliatives.

For full details regarding the removal of cerebral tumours, see p. 78.

INTRACRANIAL INFLAMMATION

Inflammation of the brain and meninges may happen either as the result of injury, or may spread inwards from inflammation of external parts, such as disease of the cranial bones, erysipelas of the scalp, etc. It is always due to organismal causes. In those cases which follow injury there is usually an open wound through which the organisms gain entrance; but meningitis may occur without any visible external wound, as the result of a bruise, the organisms in this case either being transmitted through the superficial bruised parts to the meninges, or finding their way there by the blood-vessels supplying the part.

The inflammatory processes are of different degrees of intensity; thus the meningitis may be acute, chronic, or subacute.

Probably many of those cases which simulate meningitis, and come on very quickly without any palpable external wound, are due to spreading œdema.

When organisms are introduced *directly* by a wound of the brain or meninges, the symptoms come on quickly, that is, from three to six days after the injury; where they are introduced *indirectly*, two or three weeks, or more, may pass before active inflammation is set up.

The chief indirect channels through which organisms gain entrance to the meninges are: (*a*) by the venous system—the emissary veins may become thrombosed and organisms may invade the clot and eventually reach the membranes; (*b*) by the lymphatics; (*c*) by metastasis, as in pyæmia; the organisms circulating in the blood-stream and being deposited in the part devitalised by the injury.

The position of intracranial inflammation, like that of intracranial hæmorrhage, may be subcranial, subdural, or cerebral.

In the subcranial division there is usually a localised pachymeningitis, or an abscess may form between the skull and dura mater; in the subdural form we find leptomeningitis or inflammation of the soft membranes of the brain; this form may be acute or chronic, limited or diffuse; while as a result of injury we meet with cerebral abscess and infective thrombosis of the venous sinuses.

It is necessary shortly to discuss these different forms.

1. *SUBCRANIAL OR EXTRADURAL INFLAMMATION.*—(*a*) *Simple pachymeningitis*, or inflammation of the external surface of the dura mater, is usually localised. It may result from a severe blow on the head, with or without fracture of the skull. The dura is separated from the bone over a limited area, and effusion is poured out into this cavity, but is prevented from spreading to any distance by the adhesion of the dura to the skull. The chief importance of localised pachymeningitis consists in the fact that it may cause adhesions between the dura and subjacent cortex, and by so doing cause traumatic epilepsy or persistent headache. If this condition is suspected by the persistence of headache or local pain, blisters should be applied over the painful area; and should these fail to relieve the pain, or if epileptiform symptoms appear, the bone should be trephined (see "Traumatic Epilepsy," page 99).

(*b*) *Subcranial Abscess.*—This is usually the result of a compound or punctured fracture of the skull, the dura mater being not cut through, but merely separated from the bone. Thus, a cavity forms which fills with blood-clot, and organisms attacking this sensitive medium are apt to set up suppuration.

Subcranial abscess is also frequently caused by inflammation spreading from suppurative otitis media, the roof of the tympanic cavity becoming perforated, and an abscess forming between the dura mater and the petrous bone.

The *symptoms* of subcranial abscess are local pain and tenderness on pressure, rigors, and, if the collection of pus be large, sufficient pressure may be exerted on the cortex to cause characteristic focal symptoms of spasm or paralysis.

Where there is no open wound, or where such a wound has closed before the abscess has developed, the scalp tissues over the site of the abscess are apt to become œdematous and swollen. This condition is known as "Pott's puffy tumour."

The *treatment* consists in opening the abscess, either by trephining or removing loose pieces of bone.

2. *LEPTOMENINGITIS.*—Under this heading are included the different

forms of inflammation of the subdural membranes, viz. the arachnoid and pia. As the surface of the brain is usually affected at the same time it should perhaps be called *meningo-encephalitis.*

This form of meningitis may be limited by adhesions to a small area of the brain and membranes, and may give rise to symptoms pointing to such limitation, *e.g.* an adhesion of membranes to brain may give rise to Jacksonian epilepsy, the attack starting in some particular group of muscles (see page 98).

In the large majority of cases, however, meningo-encephalitis is *diffused* over a large area of the brain surface. It may occur in various degrees of intensity.

(*a*) *Acute Leptomeningitis.*—Organisms may reach the membranes either directly through a wound of the skull and dura mater, or by one of the various channels previously mentioned. The intensity of the disease depends partly on the method of inoculation, the symptoms being more rapid and intense if there be direct contamination by open wound down to the membranes. It also depends on the virulence of the particular organisms introduced, the milder ones, such as the pneumococcus, not causing such a severe attack as the staphylococci and streptococci which are more commonly found.

There is intense hyperæmia of the inflamed surface, with small points of extravasation scattered over the surface of the pia mater. The subarachnoid space and ventricles usually are found to contain an excess of cerebro-spinal fluid which is turbid with lymph, blood, or pus, and which coagulates on being heated. The membranes are thickened and congested, the surface of the convolutions œdematous and flattened. The pia mater is covered with lymph, which tends particularly to collect in the sulci, and the brain substance itself shows evidences of softening and hyperæmia, the latter often accompanied by punctiform hæmorrhages.

Symptoms.—In most cases the first symptoms appear about thirty-six or forty-eight hours after the injury. Severe headache, with a rising temperature and a full bounding pulse, are the earliest symptoms. The patient may feel chilly or have a definite rigor. Photophobia, vomiting, muscular twitchings, delirium, and other nervous phenomena may follow, and bear witness to the intensely irritable condition of the brain.

In the course of a few hours the symptoms will have advanced considerably: the pain in the head is extremely severe, the pupils are contracted, and squinting or optic neuritis may be noticed.

The patient gradually gets weaker, worn out, and drowsy, and coma gradually supervenes. The pupils now become dilated and immobile, and other symptoms of compression of the brain by excess of cerebro-spinal fluid, such as a slow, irregular pulse, stertorous breathing, paralysis, etc., may become noticeable before death.

The meningitis is usually farther advanced at the site of injury than at other parts of the brain, the inflammatory process gradually spreading from the original site of infection (which is usually on the convexity) towards the base of the brain. The symptoms of meningitis of the base and of the convexity are somewhat different in some respects.

In *convexity meningitis* there is more liability to convulsions, and these at first may be limited to certain groups of muscles, indicating a limited involvement of some of the centres in the Rolandic area.

In *basal meningitis* optic neuritis comes on early and is intense, the head is retracted, and squinting, due to irritation or pressure on the nerves supplying the muscles of the eye, is of frequent occurrence.

Treatment.—The treatment of true septic leptomeningitis is highly unsatisfactory.

The patient should be kept in a cool, dark, and quiet room, and should not be allowed to talk. The diet should be low, and the bowels should be freely opened. The continuous application of cold to the shaven head or leeches are useful, and venesection may be tried if the patient be not too weak to stand such a line of treatment. Surgical interference in the direction of drainage by opening the subarachnoid space is probably quite useless when the disease has made any progress, still it may relieve intracranial pressure, and will, at least, do no harm. In the earliest stages the thorough cleansing and draining of any septic head wound is a part of the routine practice, but this is more of the nature of prevention than of cure.

(*b*) *Chronic leptomeningitis* is apt to occur as the result of traumatism, and is often associated in these cases with a syphilitic history. Like chronic pachymeningitis, it is accompanied by localised pain in the head, and is apt to be followed by traumatic epilepsy. Counter-irritation by blistering, and in epileptic cases trephining, are the most important forms of treatment.

Occasionally a *subacute* or *delayed* form of meningitis occurs in which the symptoms do not follow the injury till after the lapse of some weeks, or even longer. These cases do not, as a rule, run such an acute course as those first described, nor do they require such a vigorous line of treatment.

3. *Cerebral Abscess*

Cerebral abscess is met with occasionally in the frontal or parietal lobes of the brain after injury, and especially after penetrating wounds, such as gunshot wounds, which carry in foreign bodies, such as hair, etc., into the substance of the brain. Abscess may also occur as the result of contusion from direct injury or from contre-coup, the organisms spreading along the lymphatics or blood-vessels from the surface into the deeper parts, or being deposited by the blood-current in the parts weakened by injury.

In other cases the abscess may be metastatic, and secondary to some suppurative focus at a distance, as in pyæmia; or it may spread from septic osteitis, *e.g.* necrosis of the cranial bones due to syphilis, tubercle, etc.

By far the commonest cause of cerebral abscess, however, is chronic suppurative otitis media.

In those cases following injury the abscess usually runs an acute course, and has no limiting capsule. In most cases, however, which follow middle ear disease, the abscess is chronic and encapsulated. It may lie latent for months, or even for years, without causing any marked symptoms, and then, by a sudden increase in size or by bursting, may draw attention by causing compression of the brain or meningitis.

Symptoms of Cerebral Abscess.—If resulting from injury the abscess runs an acute course. On the second or third day after the wound there is severe headache, the temperature rapidly rises, rigors may take place, and symptoms which at first simulate those of meningitis are present. Compression symptoms shortly follow, and the patient, if unrelieved, dies comatose in the course of a week or ten days.

When the abscess is secondary to suppurative otitis media, or similar causes, the symptoms are somewhat different, and the case runs a subacute or chronic course.

The characteristic symptoms of a chronic cerebral abscess are *headache, drowsiness, vomiting*, a *dirty tongue*, a *subnormal temperature*, and *optic neuritis*.

(*a*) *Headache* is usually severe and sometimes excruciating, though in some cases it may be entirely absent. It is usually general at first, but subsequently is felt chiefly over one side of the head, and there may be marked tenderness on pressure or percussion over the area overlying the abscess. The pain may be constant or intermittent, occasionally coming on in severe paroxysms.

(*b*) The patient is *drowsy* and much inclined to sleep. When asked questions he may take a long time to answer, and speaks in a markedly slow and deliberate manner.

(*c*) The *digestive system* is much disturbed, as evidenced by an entire loss of appetite, constipation, and an extremely dirty tongue and foul breath.

(*d*) *Vomiting* is apt to occur without a feeling of nausea, and in no relation to meals; the patient frequently vomits immediately on waking up from sleep.

(*e*) The *temperature* is, as a rule, distinctly subnormal (96° to 97° F.) unless there is accompanying meningitis. If cerebral abscess and meningitis coexist, the latter obscures the diagnosis by causing a high temperature. This is particularly the case if the meningitis be basal.

(*f*) The *pulse* is slow and apt to intermit.

(*g*) The *respiration* in the later stages is apt to take on the Cheyne-Stokes' character.

(*h*) There is *optic neuritis* in most cases, most marked on the affected side. The pupil on the affected side is apt to be dilated and insensitive to light.

(*i*) *Epileptiform attacks* may occur.

(*j*) *Localising symptoms* may be present, due to pressure or irritation by the abscess of the sensori-motor areas.

The commonest positions in which chronic abscess occurs in the brain are the temporo-sphenoidal lobe, the cerebellum, and the frontal lobe.

Abscess in the *temporo-sphenoidal lobe* is very common after mastoid disease, and is due to an extension upwards of the suppuration through the roof of the tympanic cavity. Interference with smell or taste (for which senses the centres are situated in the temporo-sphenoidal lobe) is not often marked, as the other healthy side of the brain takes up the duties of the diseased side to a considerable extent. There may be sufficient pressure exerted by a temporo-sphenoidal abscess to cause paralysis of the muscles of the face or arm of the opposite side; and if the abscess be situated on the left side of the head the speech centre in the third frontal convolution may be affected.

Abscess in the *cerebellum* is frequently secondary to lateral sinus phlebitis with infective thrombosis. This is a commoner result of mastoid disease in adults than in children, as in the latter the mastoid cells are not fully developed till about puberty, and consequently the pus from the middle ear does not tend to spread into the mastoid prominence, and so come in close relation with the sigmoid sinus as it does in the adult.

In cerebellar abscess optic neuritis is well marked, vomiting is specially frequent, and there is usually retraction of the head.

Abscess in the *frontal lobes* is apt to show little distinctive sign except, perhaps, interference with the mental functions. If the abscess affect the third frontal convolution on the left side we may have aphasia present.

Abscesses situated in the sensori-motor area will usually denote their exact position by causing spasm or paralysis of the groups of muscles corresponding to the brain centres affected.

Diagnosis of Cerebral Abscess.—The chief conditions with which cerebral abscess may be confused are meningitis, mastoid disease, lateral sinus phlebitis, and tumour of the brain. The following are some of the main points of distinction:—

(*a*) *In meningitis* there is more evidence of irritation, *e.g.* severe pain, delirium, contraction of the pupils and photophobia, spasm, and rigidity of muscles. The symptoms of meningitis come on soon after the infection, and their onset is abrupt; the pulse and temperature are both raised.

(*b*) *In mastoid disease* there is deafness, a history of otorrhœa, with pain and œdema over the mastoid, and other local signs of the disease (*q.v.*) The difficulty in diagnosis arises from the fact that not only is cerebral abscess frequently secondary to mastoid disease, and the two conditions may coexist, but certain symptoms, such as giddiness, vomiting, and even optic neuritis (symptoms which are usually indicative of cerebral abscess), are present in a certain number of mastoid cases. That this is the case is proved by the fact that cleaning out the mastoid antrum may in such cases remove the symptoms. There is probably some local basal meningitis and extradural effusion in these cases to cause the above symptoms.

(*c*) *In lateral sinus phlebitis* with infective thrombosis the most marked symptoms are practically those of pyæmia, viz. severe rigors, sweatings, and a very high temperature with marked remissions. There is usually some tenderness over the upper part of the internal jugular vein in the neck; vomiting and optic neuritis are absent.

(*d*) *Cerebral Tumours.*—The diagnosis here is often so difficult as to be impossible by any means short of exploratory operation.

The absence of an original septic focus, and the steady, unrelenting progression of the symptoms of a tumour from bad to worse are perhaps the chief points of importance. While optic neuritis is common enough in cerebral abscess, it may be considered an almost invariable symptom of cerebral tumour. There is, as a rule, much less gastric disturbance in the case of tumour.

The diagnosis between chronic cerebral abscess and spreading œdema of the brain is not usually difficult. When the conditions occur together, as may be the case, the symptoms are confusing.

In an uncomplicated œdema, the history of a recent injury, the rapid onset of symptoms, the quick pulse, high temperature, and the absence of rigors, vomiting, and optic neuritis, are usually sufficiently indicative of the nature of the case.

Treatment.—This consists in evacuating the pus and draining the cavity. If this can be done without interference with any vital part of the brain the prognosis is very favourable.

Having determined the site of the abscess by a careful consideration of the symptoms, a trephine of at least 1 inch in diameter should be applied directly over this point. If the abscess be subcranial in position pus will at once follow the removal of the disc of bone. If subdural, the dura mater will bulge in the trephine hole. It must be carefully opened, and if the abscess be not yet reached, the surface of the brain must be carefully examined by inspection and palpation, and, if necessary, a fine exploring needle thrust into the substance of the brain.

If pus be found, a pair of sinus forceps should be gently pushed in along the needle, and a rubber drainage tube of about ½ inch lumen passed into the abscess cavity. The contents should then be *gently* washed out, and the end of the tube brought out through a dependent angle of the wound. (For fuller details see p. 78.)

In *frontal* abscess secondary to disease of the nasal bones, trephine at least 1 inch above the upper margin of the eyebrow so as to avoid opening the frontal sinus.

In *temporo-sphenoidal* abscess trephine ½ inch behind and 1 inch above the centre of the external auditory meatus.

In *cerebellar* abscess trephine 1½ inches behind and ½ inch below the centre of the external auditory meatus, so as to avoid the lateral sinus.

If there be a doubt as to whether the abscess be situated in the cerebellum or temporo-sphenoidal lobe, it has been recommended to trephine 1½ inches behind and ½ inch above the centre of the external auditory meatus. Explore the temporo-sphenoidal lobe first, and if you fail to find pus there, remove bone with the gouge forceps downwards and backwards from your original trephine opening and expose the cerebellum. In this way both the temporo-sphenoidal lobe and the cerebellum may be readily examined through one opening in the skull.

The chief apparent objection to this suggestion is the risk of contaminating the meninges above the tentorium with the pus coming from a cerebellar abscess below the tentorium. It is safer to make two distinct openings, the first of which can be closed and sealed with collodion before the second is made.

4. *Infective Thrombosis of the Venous Sinuses.*—Thrombosis of the sinuses of the dura mater occasionally takes place in cases of marasmus. The longitudinal sinus is the one most commonly affected in this way. The form of thrombosis, however, which solely interests the surgeon is that which is due to infection by organisms, and is met with in connection with local inflammation, the result of injury, septic osteitis of the cranial or facial bones, facial erysipelas or cellulitis, and, particularly, suppurative otitis media. The micro-organisms usually reach the sinus by the infection spreading along thrombosed emissary veins from the septic focus outside the cranium to the intracranial sinuses.

The most common sinus affected is the lateral sinus. This is usually the result of otitis media. The longitudinal sinus may be thrombosed from compound fractures of the skull or septic scalp wounds.

The cavernous sinus is affected in cases where the nasal fossæ, mouth, or orbit are the seat of inflammatory septic mischief.

Owing to the invasion of micro-organisms a clot forms in the lumen of the sinus. This in turn is invaded by organisms, tends to break down and to be carried into the general blood-stream, giving rise to symptoms of pyæmia, while infarcts in the lung, followed by pneumonia or gangrene of the lung or metastatic abscesses in other parts of the body, may result. In the case of thrombosis of the lateral sinus, basal meningitis and cerebellar abscess are frequent consequences.

Symptoms.—In all cases of septic thrombosis of the intracranial sinuses and the prominent symptoms are those of a pyæmic character. Thus we meet with high fever with marked remissions, the temperature ranging from 103° to 106° F., and dropping suddenly to 96° or 97° F. The pulse is rapid and weak. Rigors are frequent and severe, and are a very characteristic symptom.

In addition to these symptoms we may have vomiting, sweating, headache, severe pain over the affected sinus, and, in addition, there may be evidences of lung mischief, metastatic abscess in joints or elsewhere, and other signs of septic infection.

In the case of the cavernous sinus there is protrusion of the eyeballs and œdema of the lids, due to pressure on, or thrombosis of, the veins of the orbit, while pressure on the nerves may give rise to ptosis or squinting.

In the case of the lateral sinus there will usually be clear evidence of a previous otitis media followed by symptoms of mastoid disease, viz. œdema and pain over the prominence.

The internal jugular vein in the neck may, in the later stages, be palpably resistant and tender to pressure, while the deeper glands situated in relation to the upper end of the sterno-mastoid muscle may be inflamed, and may suppurate.

Treatment.—Early and energetic treatment is necessary, as otherwise the results of lateral sinus phlebitis are very serious. The condition of the middle ear should be attended to.

In case of mastoid disease thorough clearing out and drainage of the mastoid antrum and cells should in all cases be done, and the bone carefully chiselled away backwards till the sigmoid sinus is reached at a point ¾ of an inch directly behind the external auditory meatus.

Pus will usually be found surrounding the lateral sinus. This should be washed away and the sinus examined. If it appear to be resistant, if the mastoid vein be blocked, and if on pricking the lateral sinus with a fine needle no blood come out, thrombosis is present, and the following operation should be done:—

The internal jugular vein should be exposed in the upper part of the neck by an incision along the inner border of the sterno-mastoid muscle. The vein should be ligatured so as to prevent general infection by the septic contents of the lateral sinus being carried down the jugular vein towards the heart. The lateral sinus should then be opened, the contents washed or gently scraped out, and the wounds plugged with gauze.

In the case of infective thrombosis of the longitudinal or cavernous sinuses the treatment is chiefly preventive, and consists in dealing with the cause of infection by the careful cleansing of scalp wounds, the removal of pieces of dead bone in cases of compound fracture and necrosis, and by attending to ordinary surgical indications.

EPILEPSY

As surgical interference has proved futile in cases of ordinary or idiopathic epilepsy, while its results in other forms of the disease are very encouraging, it is necessary to discuss shortly the varieties met with.

Idiopathic epilepsy is distinguished by the attack coming on suddenly, with little or no warning, except, in many cases, the occurrence of a cry; the patient falls to the ground, loses consciousness, and is seized by general convulsions which last a few minutes. Foaming at the mouth and biting of the tongue are familiar symptoms. Deep unconsciousness follows the convulsive attack, and subsequently the patient passes into a deep sleep which often lasts several hours.

In these, the common cases of epilepsy, surgical operation is useless, although temporary cessation of the fits has occasionally been noticed to follow trephining.

JACKSONIAN EPILEPSY.—In this variety there is a conscious sensation of the commencement of the attack in some region of the body, *e.g.* the arm, leg, or face, and the convulsions, beginning in such a part, spread from it in a definite order up or down the body. The patient does not, as a rule, lose consciousness, though he may do so if the convulsions eventually become general.

This form of epilepsy, as shown by Hughlings Jackson, is due to organic disease affecting some part of the sensori-motor area of the brain cortex; and it has been noticed that such an organic lesion will be always found in that part of the brain cortex which corresponds to the muscles which are first to be convulsed, while the extent to which such convulsions spread to neighbouring parts depends upon the severity of the initial irritation—in other words, the slighter the cortical irritation the more localised will be the convulsions and *vice versâ*.

There is often a feeling of considerable weakness left for a time in the muscles chiefly involved in an attack of Jacksonian epilepsy.

SENSORY EPILEPSY is the term applied when the centres corresponding to the organs of special sense are involved by disease. Thus the senses of sight and hearing, taste and smell, are occasionally affected, and if an attack of epilepsy begin by a perversion or hallucination referred to one of these senses, we may be guided to a correct diagnosis of the position of the initial lesion—for example, a flash of light or hallucination of sight points to the occipital lobe; a perversion of taste or smell to the tip of the temporo-sphenoidal lobe; a sound to the temporal convolutions.

In like manner an irritation of the third frontal convolution of the left hemisphere (which in right-handed people is the motor speech area) may be followed by an arrest of function of speech, this constituting the *aphasic* form of epilepsy, while a lesion in the frontal lobes may be evidenced by temporary interference with the *mental* functions.

TRAUMATIC EPILEPSY.—A considerable number of cases of Jacksonian epilepsy are referable to an injury. There are certain points to be borne in mind in this connection, viz. that the epilepsy may not show itself for a long time—several years, it may be, after the infliction of the injury; that the apparent severity of the trauma has not necessarily any relation to the severity of the fits; that a hereditary tendency to epilepsy is frequently present in those who develop epileptic symptoms after injuries to the head, and it seems to act as a predisposing cause.

In a few cases a mere injury to *the soft parts* covering the skull seems capable of causing the fits, pressure on the resulting cicatrix occasionally giving rise to a seizure by reflex action.

The *skull* is frequently the site of the lesion, a depressed fracture, particularly in the shape of a sharp spicule of the inner table, irritating the subjacent brain. (This fact appears to indicate the propriety of elevating depressed bone in recent cases of fracture, even if no signs of compression of the brain are present.)

The *dura mater* is often thickened and adherent to the skull or membranes beneath, and these in their turn may be altered and adherent to the brain.

The *brain* itself may show evidences of injury, such as cysts (the result of hæmorrhages), patches of softening, sclerosis, or local cerebritis.

While the majority of cases of Jacksonian epilepsy with localising symptoms is due to traumatism, other causes are occasionally met with, *e.g.* tumours of different kinds growing from the skull, membranes, or brain; cysts and other alterations in the brain or membranes, the result of embolism or thrombosis in the blood-vessels, etc.

Treatment.—The surgeon having determined that he has to deal with a case of epilepsy suitable for operation, frequently finds a difficulty in deciding at what point to open the skull, as the localising symptoms may not point to that part of the cortex which lies underneath the site of the injury. "When, however, the injury and the localising symptoms do not coincide, it is better to follow the localising symptoms rather than the surgical injury" (Allen Starr).

The chief indication in operative interference is, of course, to remove the cause of irritation where that is possible.

Where the symptoms depend on a depression or outgrowth from the bone, or an alteration in the dura mater, the prospects of recovery are extremely good; where, however, the brain itself is at fault the results of operation are not encouraging.

In some cases the cortex is visibly altered in colour and appearance, and if this area corresponds to the localising symptoms, it is recommended by some surgeons to excise the patch of cortex so altered. In some cases no such alteration can be found to correspond to the symptoms, and in these cases Horsley recommends the use of a mild faradic current, by which the exact area corresponding to the convulsed muscles may be discovered with a view to its removal. While this proceeding may put an end to the fits for a time, it necessarily causes paralysis of the muscles concerned, and in the course of time the cicatrix resulting from the operation is apt by its contraction to set up a renewed condition of irritation with return of the fits.

The prognosis of cases of epilepsy which are not operated on being extremely unfavourable, and the dangers of an exploratory operation being very small, it is wise to recommend surgical interference, as it is not often possible to make a definite diagnosis till the skull is opened. While the large majority of cases are much benefited for a time by operation, in a large proportion (especially where the focus of irritation cannot be removed without damaging the cortex) the fits recur after a time; a guarded prognosis should always accordingly be given. If operation be thought advisable, it should not be too long delayed, as it is important not to allow the epileptic "habit" to become confirmed.

HERNIA CEREBRI

When a protrusion of brain takes place through an *acquired* opening in the skull it is termed a *hernia cerebri*. (Protrusion of the brain or its membranes through a *congenital* defect in the skull is met with in meningocele, encephalocele, etc.)

In every case hernia cerebri is caused by increased intracranial pressure forcing the brain through the abnormal opening in the skull and dura; for even when such an opening exists, if there be no increased intracranial pressure, herniation will not take place.

The brain will not protrude unless the dura mater is wounded as well as the skull. The condition is usually found either in connection with compound fractures of the skull, syphilitic necrosis, or after surgical operations.

The necessary factor of abnormally great intracranial pressure is produced by one of the following:—

(*a*) Intracranial hæmorrhage.
(*b*) Œdema of the brain.
(*c*) Tumour of the brain.
(*d*) Inflammatory swelling of cranial contents.

Of these the last is by far the commonest. It is very apt to ensue as the result of septic wounds of the brain, accidental or surgical, and is a not uncommon sequela of compound fractures of the skull in which asepsis has not been obtained. In some cases a definite localised abscess is found in the brain substance beneath the hernial tumour.

Again, in operating on a tumour of the brain the surgeon may either be unable to remove the growth, and a hernia cerebri may follow the operation (a result which is highly beneficial and desirable in most instances, as thereby relief is afforded to the intense intracranial pressure), or as a result of his interference with the brain a spreading œdema may be set up, which increases the intracranial pressure in such a way as to cause a hernia cerebri.

In a few cases of depressed fracture of the skull it happens that an effusion of blood (assisted, perhaps, by depressed bone) may produce sufficient pressure to cause hernia; and this form of the disease may appear immediately after the injury. Usually some days, or even weeks, elapse between the injury to the skull and the formation of the hernia. The portion of brain protruded generally escapes through a small opening in the skull; for if there were a large opening in the skull, the intracranial pressure would probably have been sufficiently relieved to prevent the formation of a hernia.

The herniated brain tissue—at first red, turgid, and pulsating—becomes œdematous and swollen, and eventually disorganised, or even sloughing on its surface, these changes being due to the pressure on the vessels in the neck of the tumour by the margins of the opening through which it has escaped.

The *prognosis* of hernia cerebri is unfavourable: only about one-fourth of all cases recover; most of these recoveries are in cases of hæmorrhagic or bony pressure which has been relieved by operation.

Treatment.—The most important indication is to prevent the formation of hernia by taking any steps that may be possible to relieve intracranial pressure, and to be scrupulously careful about such matters as asepsis and drainage.

When a hernia has once formed no very active treatment of the tumour is of any use; indeed, it is usually mischievous. It is evident that as long as the intracranial pressure remains our chief object should be to deal with it rather than with the hernia. It has been suggested to apply pressure to the mass by means of elastic bandages, or to treat it with astringents, alcohol, etc., or even to shave it off. All these plans are of very doubtful value. If the intracranial pressure be lessened by the absorption, let us say, of some tuberculous deposit at the base of the brain, the hernia will recede of its own accord and the wound will close. If, on the contrary, the intracranial pressure increases, the most sensible line of treatment is to considerably enlarge the opening in the skull through which the hernia has come in order to relieve general pressure on the brain, and possibly to minimise injury to the protruded cortex.

CEPHALOCELE

It occasionally happens that the membranous layer which goes to form the skull of the newly-born infant is imperfectly developed; that ossification in the cranial bones is consequently incomplete; this gives rise to a protrusion of the membranes and other cranial contents at those portions of the skull which offer least resistance. In some cases the presence of intrauterine hydrocephalus seems to be partly or wholly responsible for the condition.

These protrusions usually take place in the middle line of the head, the commonest position being posteriorly, in connection with the occipital bone, the tumour projecting through an opening which runs from the foramen magnum up to the occipital protuberance, called by Sutton the "temporary occipital fontanelle."

Next in frequency they are found at the root of the nose in relation to the fronto-nasal suture; but they are also met with occasionally elsewhere in connection with the sutures of the skull, sometimes at its lateral aspects, and may project into the orbit, mouth, or nose, in which latter position they have been mistaken for polypi.

There are three varieties of the condition depending on the contents of the protrusion.

1. *Meningocele*, in which there is a protrusion of the membranes containing cerebro-spinal fluid. The tumour is usually somewhat small, globular in shape, sessile or more rarely pedunculated; more or less translucent; fluctuating.

The skin covering the tumour may be normal in appearance, or thin and of a bluish colour, and covered with dilated blood-vessels. The tumour becomes tense on forced expiration, such as in coughing or crying, and the contained fluid can be returned by pressure more or less completely inside the cranium, when the margins of the opening in the bone may be distinguished.

2. *Encephalocele*, where in addition to the membranes there is brain substance in the protrusion. These tumours are opaque; they do not fluctuate, but frequently pulsate synchronously with the brain. They are usually small, with a broad base; are most commonly situated at the back of the head, and attempts to reduce them are followed by convulsions or other symptoms of brain pressure.

3. *Hydrencephalocele*, where in addition to the membranes there is a protrusion of brain substance which is distended by a prolongation into the tumour of one of the ventricles containing ventricular fluid.

In hydrencephaloceles occurring in the frontal region, laterally, or through the upper half of the occipital bone, the dilated lateral ventricle will be found in the sac; where the lower part of the occipital bone is deficient, the cerebellum and the distended fourth ventricle are the parts protruded.

The brain substance in these cases is much thinned and altered in appearance, and a layer of fluid is sometimes found between the membranes and the brain in addition to the fluid inside the distended ventricle.

These are the largest, the most frequent, and the most fatal of the cephaloceles. They are generally at the back of the head, and pedunculated; they fluctuate; they do not usually pulsate; are more or less translucent; become tense on forced expiration; and are irreducible.

Diagnosis.—A consideration of the above-noted characteristics of these tumours, namely, their position, shape, reducibility, pulsation, their increase on coughing or crying, and the existence of an abnormal aperture in the skull, will usually distinguish them from such things as dermoid cysts, nævoid tumours, or cephalhæmatomata. The most difficult case to determine is that of a tumour lying at the inner angle of the eye, or near the anterior fontanelle—a common situation for dermoid cysts, as it is for meningoceles. The introduction of a small sterilised exploring needle will settle the point, as in the case of the meningocele clear cerebro-spinal fluid will be drawn off, whereas the contents of a dermoid cyst are semi-solid and grumous, and will probably not come through the needle.

Prognosis.—The outlook in the majority of cases is very unfavourable. Death not infrequently takes place shortly before or soon after birth, and in the case at least of encephaloceles and hydrencephaloceles there is no tendency to cure, and little chance of the child living many months. The usual cause of death is inflammation occurring first in the skin and causing its destruction, and then spreading to the tumour contents. A certain number of cases of pure meningocele are supposed to be eventually shut off from the cranial cavity by closure of the neck of the sac; but it is not possible to say that many of such cases are not congenital cysts.

Treatment.—In most cases it is advisable not to adopt any active treatment.

Protect the tumour from injury by a suitable apparatus—something on the principle of a vaccination shield answers sometimes—keep the skin over the tumour clean and dry so as to lessen chances of irritation.

In cases of simple meningocele two plans have been recommended.

(*a*) *Puncture.*—With a fine aseptic needle draw off some of the fluid from the sac, and then apply gentle pressure, or inject a small quantity (ʒss. to ʒj.) of Morton's fluid (iodine, gr. 10; iodide of potassium, gr. 30; glycerin, ℥j.), or else, and more safely,

(*b*) *Aseptic removal of the sac* after ligature of its neck, and subsequent closure of the hole in the bone, where that is large, by an osteoplastic operation.

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Breast. *See* MAMMARY GLAND.

Breath.—The expired air which is popularly known as the breath consists of atmospheric air returned from the lungs saturated with watery vapour, and otherwise altered chemically and physically by having increased its proportion of carbonic acid gas, having become warmer, and being charged with more or less putrescible organic matter which in certain diseases gives it an offensive odour, as in some forms of dyspepsia, in ozæna, tonsillitis, bronchiectasis, phthisis, pulmonary gangrene, and typhus fever. It normally contains 4·782 vols. per cent less oxygen and 4·38 vols. per cent more CO_2 than ordinary air. These figures give the respiratory quotient as $\frac{CO_2}{O_2} = \frac{4{\cdot}38}{4{\cdot}782} = 0{\cdot}905$, but this may vary under normal conditions. These chemical changes are the immediate result of the exchange of gases in the lungs, and remotely depend upon the vital exchange which takes place in the tissues (*vide* "Respiration"). Expired air also contains traces of ammonia from the blood and traces of H and CH_4, probably derived from the digestive tract, whence also come such products of decomposition as acetone, which gives a peculiar and characteristic odour to the breath. This odour has been most often observed in diabetes, but as acetone is found in the urine after taking alcohol, in pneumonia, measles, and other acute diseases, especially in children, it is unsafe to attach to it any great diagnostic value, but undoubtedly its presence would suggest the desirability of a careful examination of the urine.

Sulphuretted hydrogen, if formed in the stomach or small intestine, is excreted through the lungs as are the vegetable volatile sulphides found in onions and garlic. Foul-smelling breath is perhaps most often dependent upon putrefactive processes in carious teeth or among the papillæ of a coated tongue. The normal temperature of the breath is 36·3 C., which is very near the temperature of the body. External cold increases the intake of oxygen and the discharge of carbon dioxide, while a rise of external temperature is followed by a diminution of both. Muscular exercise acts like cold, and undoubtedly a considerable part of the effect of cold is due to the involuntary movements excited by it.

Food produces the same result, while fasting lowers it. In the case of Cetti, the fasting man, the absorption of oxygen and discharge of carbon dioxide per kilo. of body weight fell rapidly, so that the respiratory quotient which before the fast was 0·73, on the third day of fasting had fallen to 0·65, and remained during the remainder of the fast between 0·65 and 0·68. Vegetable food raises the respiratory quotient to nearly unity, while on flesh diet it is about 0·74, and on a mixed diet somewhat higher, these differences depending upon the amount of carbon present in the food.

The relations of the breath to the etiology of disease are not perfectly clear, but it is known: (*a*) that animals made to rebreathe the same air die ultimately of asphyxia; and (*b*) there is a considerable volume of evidence to show that persons living in crowded and ill-ventilated rooms, where they are compelled to breathe over again air which has been already exhausted by themselves and others, become anæmic and ill, and are especially liable to become attacked by tuberculosis; (*c*) the tubercle bacillus has been found in the expired breath of consumptive patients, so that doubtless it may afford a channel of infection for this and for other diseases. It is believed that measles, scarlatina, diphtheria, influenza, and the infection of a common cold, may be communicated by the breath—a matter of the greatest importance in reference to prophylaxis.

A good deal of discussion has taken place as to the causes of the asphyxiating property of air vitiated by respiration, and the subject is considered in the article "Asphyxia."

Clinical Diagnosis.—Examination of the breath may be utilised for purposes of diagnosis:—

(*a*) For the determination of death it is a common practice to apply a feather to the mouth or the nose, or the polished surface of a mirror, in order to see whether any movement of air or deposition of watery vapour takes place.

(*b*) The temperature of the breath varies with the body temperature, for example, falling very low in the cold stage of cholera and rising high in fever.

(*c*) The odour of the breath is modified by smoking or chewing various aromatic substances, or by eating certain articles of food, such as garlic, which contain strong smelling compounds. Alcohol gives a characteristic odour to the breath, as do many substances used as drugs, for example, bismuth, copaiba, and the mineral poisons, especially mercury. The breath of women during menstruation may have a sweet odour like that of chloroform. In uræmia the breath often smells of ammonia, which may be demonstrated by the production of thick white fumes when a glass rod dipped in hydrochloric acid is brought near the mouth, while in diabetes its odour has been variously compared to hay, apples, sour beer, and vinegar. In pyæmia and allied conditions (septicæmia, etc.) the odour of the breath is sweet. In dyspepsia and constipation the breath is foul and sometimes almost fæcal in odour, while, when stercoraceous vomiting is present, it is undoubtedly fæcal. Local morbid conditions, such as dirty or decayed teeth, ozæna, chronic follicular tonsillitis, catarrh of the mouth and tongue, ulceration or gangrene, malignant disease, caries of bone in the mouth or nose, ulceration of the larynx or decomposition of retained bronchial secretion in dilated bronchial cavities, or gangrene of the lung, may give rise to the most foul odours of decomposition in the breath.

(*d*) Microscopical examination of the expired air has in phthisis shown the presence of the tubercle bacillus (A. Ransome), and if applied in other diseases might succeed in demonstrating other pathogenic bacteria, but this method has not so far been very extensively used. Many authorities consider that the tubercle bacillus is never found in the breath in ordinary respirations, but only when associated with the act of coughing.

(*e*) The same may be said for bacteriological examination, this mode of searching for microbes not having been systematically applied to the breath.

(*f*) Chemical examination is the only scientific method which has hitherto yielded much result. The respiratory quotient is found to vary according to the state of metabolism of the body; in inanition it is diminished, while in certain wasting diseases it is increased. There is said to be an increase of CO_2 in asthma and bronchitis, while in cholera there is a decrease.

In Bright's disease and uræmia the ammonia is greatly increased. Marsh-gas may be expired in such quantity as to make the breath inflammable, and several instances have been recorded where the patient has accidentally set fire to his breath, for instance, when trying to light his pipe. Such patients have generally suffered from dilated stomachs, in which decomposition of food was associated with the formation of this gas.

In diabetics the breath sometimes contains acetone. In poisoning from hydrocyanic acid this acid is present in the expired air.

Treatment.—As evil-smelling breath is generally a secondary symptom, its treatment depends for the most part upon the removal or cure of the primary condition, and it would be out of place here to do more than indicate the lines of treatment. Where the seat of the disease is in the mouth it is accessible, and carious teeth, etc., should be stopped or removed, a coated tongue scraped or disinfected, the pharynx sprayed with an antiseptic solution, etc. For ozæna the use twice a day of a nasal douche is often the only remedy.

In acute diseases, where the mouth is foul with sordes, it should be cleaned with glycerine and rose water. The fœtid odour from bronchiectatic cavities and gangrene of the lung may be kept down by creasote taken in capsules. Dr. G. Vivian Poore has recommended garlic for this purpose in the belief that this vegetable contains an antiseptic principle to which it owes its reputation as an article of diet. In two cases of the kind wherein a trial was made of Dr. Poore's suggestion, the stench was diminished, and was curiously enough not replaced by any offensive smell of garlic.

Putrefactive changes in a dilated stomach can be most efficiently treated with lavage by means of a soft stomach tube and a tepid solution of salicylate of soda (1-1000).

The value of hot intestinal irrigation in uræmia and similar diseases has yet to be determined, but is at least worthy of a fair trial.

Bright's Disease. *See* NEPHRITIS.

Broad Ligament, Diseases of the.

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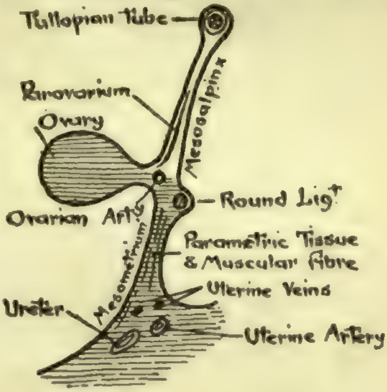
    MESOSALPINX
    MESOMETRIUM

THE serous fold known as the broad ligament is divided into the mesosalpinx or mesentery of the Fallopian tube and the mesometrium proper, which lies below the level of the attachment of the ovary. In disease the mesosalpinx is mainly associated with tumours, the mesometrium with inflammatory deposits.

Displacement of the Folds of the Broad Ligament by Tumours.—Parovarian cysts, cystic tumours developed in the hilum of the ovary and, in rare instances, tumours of the ovary proper, make for themselves a capsule of the mesosalpinx, which they greatly distend. This condition is easily recognised during operation by the position of the Fallopian tube, which is much elongated and stretched over the top of the capsule. These same tumours sometimes force apart the layers of the mesometrium as well; papillomatous cysts especially tend to do so. Ovarian cysts occasionally burrow into the mesometrium alone, then, if they grow forwards, the short anterior fold (see Sketch) is soon passed, and the tumour comes in contact with the anterior pelvic walls and the abdominal parietes, stripping up the parietal peritoneum. Ovarian cysts burrowing backwards may ultimately become retro-peritoneal. Fibro-myoma of the mesometrium often spares the mesosalpinx even when the tumour attains a great bulk. A true uterine "fibroid" may burrow into the mesometrium alone, or into the mesosalpinx as well.

INFLAMMATION.—The *mesosalpinx* is always more or less affected in perimetritis, adhering to adjacent parts. Sometimes its posterior surface becomes firmly attached to a small ovarian cyst which stretches it as it grows. This gives an appearance as though the tumour lay between the folds of the mesosalpinx, especially when inflammatory deposit on the surface of the cyst is taken for part of the imaginary capsule. An inflamed and dilated Fallopian tube opens up the mesosalpinx till the former comes in contact with the ovary. An inflamed and thickened mesosalpinx does not always bear the ligature well. This is one reason why the pedicle, after the removal of an inflamed tube and ovary, sometimes inflames, the patient suffering as much as before the operation.

Inflammation of the *mesometrium* is the chief feature in pelvic cellulitis (parametritis). Whilst the mesosalpinx contains very little connective tissue, a thick wedge of that tissue enters the lower and outer limits of the mesometrium where the folds part, becoming thinned till it ends at about the level of the ovary. It contains fat and muscular fibre. When inflamed it becomes very firm, owing to infiltration with serum. On vaginal exploration the base of the inflamed ligament feels like a metal bar running across the lateral fornix from the bony pelvis to the cervix uteri. The ureter runs along the base of the mesometrium, hence it may be obstructed by the inflammatory exudation. This is most usual when the inflammation develops around a cancerous growth advancing from the uterus; the malignant deposit itself may produce the same effect. The symptoms and course of pelvic cellulitis are described elsewhere (see "Pelvis"). The chief feature of importance in relation to this disease as affecting the broad ligament is the direction in which the inflammatory process may pass from the connective tissue of the mesometrium along similar tissue beyond its limits. It may advance forwards and upwards. This is the usual course, for the anterior fold of the broad ligament does not pass so low down as the posterior (see Sketch). Hence the serous or purulent exudation, in close relation, from the first, with the connective tissue in the forepart of the pelvis, rises in front of the peritoneum till it comes to lie behind the abdominal walls in the groin. It sometimes tracks along the round ligament into the inguinal canal. The exudation may pass backwards, sometimes creeping out of the great sciatic notch. Less frequently it tracks outwards, passing along the connective tissue of the side of the pelvic cavity, and thence often upwards into the iliac fossa. The exudation sometimes passes upwards and outwards along the infundibulo-pelvic ligament, the serous fold made by the ovarian vessels as they pass from the pelvic brim into the folds of the broad ligament. The connective tissue around the vessels is loose [and abundant. In cellulitis, after ovariotomy and allied operations, the infundibulo-pelvic ligament is a great highway for septic exudations, which may reach the loin. The lateral relations of the broad ligament explain the close association observed in many cases between pelvic cellulitis or pelvic peritonitis and inflammation in the neighbourhood of the vermiform appendix, so that it is not always clear which was the primary disease.



Abscess.—Pelvic abscess is specially described under its proper heading. The relations of the connective tissue of the broad ligament to the same tissue in its neighbourhood, above noted, should always be remembered when this affection is suspected. Abscess of the broad ligament is usually a result of pelvic cellulitis, including the form which appears after the removal of a tumour of the ovary or of old inflamed appendages, when the ligature is septic or the ligatured tissues unhealthy. Abscess of the broad ligament may also be due to infection of a hæmatoma, tumour, or fœtal sac.

Hæmatoma.—This subject is also specially treated under "Pelvis" (*q.v.*). Here we need only say that in ovariotomy the pedicle needle sometimes wounds a vein, and a hæmatoma forms in the mesometrium. As a rule this accident is speedily manifested before the operation is ended, a tense, livid blue tumour appearing below the ligature-groove. If the blood escapes slowly but steadily it may cause the ligature to yield in a few hours, or the hæmatoma may burst. Fatal intraperitoneal hæmorrhage is in either case very probable. Should the hæmatoma remain stationary, it is not likely to suppurate. The hæmatoma associated with *Extra-uterine Pregnancy* is described under that heading.

TUMOURS OF THE BROAD LIGAMENT. (1) *Of the Mesosalpinx.*—When the uterus is the seat of a large fibro-myoma, and in certain other conditions involving obstruction to the lymphatics of the pelvic viscera, large yellow blebs or bullæ of irregular form are often seen covering the mesosalpinx. These "lacunar cysts" or "subserous hygromas" do not in themselves endanger the patient, nor do they interfere much with operative manipulations; they are simply important as usually indicating serious complications elsewhere.

Anatomically the simple parovarian cyst is a true tumour of the mesosalpinx, and so are all papillomatous cysts developed from the parovarium;

and from that portion of Gartner's duct which lies within its folds. The different ways in which the folds of the mesosalpinx may be opened up by tumours from other parts is described above.

Small fatty tumours, pedunculated or sessile, have been detected in the mesosalpinx, which often contains a little fat. Parona's case of lipoma of the tube was possibly a fatty tumour of the mesosalpinx surrounding the tube.

(2) *Of the Mesometrium.*—This part of the broad ligament may be the seat of a *fibroma* or *fibro-myoma* developed quite independently of the uterus, from the fibrous and muscular tissue in its folds. A tumour of this class may attain a great size, and press upon the vessels and nerves of the pelvis and on the ureters. No other kind of abdominal tumour displaces peritoneum more widely or more freely. In comparison with uterine "fibroids," these mesometric fibro-myomas develop in younger subjects, large growths of this class having been removed from women under thirty years of age.

"Fibroids" originating in the uterus, especially near or in the cervix, tend to burrow into the mesometrium. They then lie very close to the ureter. In exceptional cases that duct has been found passing over and not under the burrowing tumour.

There is often much fat in the mesometrium, and hence it is not surprising that *lipoma* of that fold has been recorded. *Sarcoma* has also been observed. Freund describes a case of extensive invasion of the broad ligament with *echinococci.*

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Bromidiosis. *See* SWEAT GLANDS.

Bromism.—The term bromism is applied to the series of symptoms that are met with resulting from the excessive use of one or more of the bromine compounds. Since the discovery of the sedative influence of the bromides on the central nervous system, these remedies have been very extensively used for many nervous disorders, and it is specially in chronic nervous cases, *e.g.* epilepsy, that the untoward effects of the bromides are seen. The degree of tolerance for bromides varies, as for other medicinal remedies. In some cases the continued use of even small doses—5 to 10 grains—leads to a manifestation of the minor or major symptoms of bromism, while in other cases very large doses are tolerated even for lengthened periods. Clouston records an interesting case (vol. i. p. 79) of a boy of eleven tolerating 60 grains daily for two years. The nature of the disease and the idiosyncrasy of the patient are the important modifying factors.

The depressing influence of the bromides on nerve and muscle cells, taken with their irritating property during excretion by the usual channels, is responsible for the symptoms of bromism. These may be briefly summarised as follows:—

Neuro-muscular System.—A diminished capability for physical or mental work, with occasional headache, which may be of a severe character and chiefly in the frontal region, are the earlier symptoms. Later, slight forget-fulness, diminished mental capacity, general weakness of mental faculties, diminution of the palatal and pharyngeal reflex, and in still later stages some disturbance of gait may be present. In not a few cases, however, especially in epileptics, the first symptoms are those of an increased excitability of the brain cortex, amounting even to mania (Bromomania). The development of any such symptom in these cases should therefore lead the practitioner to a careful survey as to the doses and duration of the treatment.

Cardio-vascular System.—Along with the general muscular depression there is more or less evidence of general cardiac enfeeblement. The heart's action is weak and the pulse feeble. The rate may be slightly diminished or increased. A few cases are recorded where death has been attributed to the stoppage of the heart in diastole as the result of excessive use of the drug. Other indications of vasomotor disturbance may be present, *e.g.* cold extremities and clammy state of the skin.

Cutaneous System.—An acne eruption, most marked on the forehead, neck, face, and trunk, is frequently the earliest symptom to attract attention. In other cases the eruption may be erythematous, scarlatiform, pemphigoid, or even rupial in nature. In children the eruption may pass from a discrete acne type to take on a papillomatous appearance.

Other Systems.—The more important symptoms encountered in the other systems are loss of appetite, foul breath, anæmia, dilatation of the pupil, diminution of the respiratory rate, and slightly depressed temperature.

Treatment.—Prophylaxis is all-important. A full recognition of the pharmacological actions and careful observation of patients using these remedies, noting especially the condition of the skin, mucous membrane, and general vital capacity, will suffice in all cases to prevent the development of the condition in any great degree.

Curative treatment resolves itself into a stoppage of the use of the drug, strict attention to the action of the skin, kidneys, and bowels, and the use of general tonic remedies. Hydro-therapeutic measures are of great assistance, both from their local and general effects. Small doses of arsenic given in combination with the bromides are of some service in diminishing the tendency to bromism. Liq. arsenicalis is the form most commonly used, but some writers believe that arseniate of soda is the most useful preparation, being less likely to produce any gastric intestinal irritation.

Cannabis indica is also frequently used along with the bromides with the object of diminishing the tendency to the production of untoward disturbances of the nervous system.

Bronchi, Diseases of.—The affections of the bronchi and associated glands are described in the following articles:—

1. **Bronchial Glands.**
2. **Bronchitis** (for Capillary Bronchitis see "Pneumonia").
3. **Bronchiectasis.**
4. **New Growths.** See "Lungs" and "Mediastinum."

Bronchial Glands

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ANATOMY.—The glands may be classified into three main groups (Baréty):

(i.) Those around the lower end of the trachea and the main bronchi—the peri-tracheo-bronchial group.

(ii.) Those between the main bronchi, chiefly beneath them—the inter-tracheo-bronchial group.

(iii.) Those between the divisions of the bronchi—the inter-bronchial group.

These three groups are in connection with—

(i.) The superficial and deep lymphatics of the lungs;

(ii.) The lymphatics of the visceral pleura;

(iii.) Some of the lymphatics of the œsophagus.

These lymphatics communicate freely.

The glands themselves vary a good deal in size and shape, and the inter-bronchial group extend for some distance into the substance of the lung, according to Cruveilhier, as far as the fourth divisions of the bronchi. The right bronchus is shorter than the left, and is more directly a continuation of the trachea than is the left, and there are a larger number of glands in connection with the right than with the left bronchus.

From the position of the glands it will be seen that they come into relation with the following intrathoracic structures:—

The trachea, the bronchi as far as their fourth division, the lungs, the pulmonary arteries and veins, the superior vena cava, the innominate, subclavian, and azygos veins, and the aorta; the vagus nerve and its recurrent branches, and the pulmonary plexuses, the pericardium, and the œsophagus.

The tracheal bifurcation corresponds in level to a horizontal line drawn through the junction of the manubrium and body of the sternum; this corresponds behind to the spine of the fourth dorsal vertebra. The horizontal level at the point of intersection of the lines corresponding to the spines of the scapulæ will also give us the level of the tracheal bifurcation.

Morbid Anatomy and Pathology.—The bronchial glands may be the seat of various morbid changes, either as a primary condition, or secondary to changes in the neighbouring parts from which the lymphatics pass to the glands, or as part of some general condition affecting the lymphatic glands.

The most common morbid condition is *pigmentation*, and in adults this condition is practically constant. Next to pigmentation, *hyperæmia* and *inflammatory* swelling of the glands are the most common morbid conditions, and are met with in association with similar conditions in the pleura, lungs, and bronchi. The amount of swelling may be considerable, the glands being three or four times their normal size, pinkish gray in colour and succulent, or, in cases where the swelling is marked, becoming pale gray and medullary in appearance. The enlargement is not so important as the accompanying lowered activity of the glands, which results in diminished resistance to invasion of the tissues by micro-organisms, more especially by the bacillus tuberculosis. It is quite probable that owing to the retardation of the lymph stream in the lungs or pleura, the bacilli have a longer opportunity of getting into situations suitable for their growth and multiplication.

In this way the frequency of tuberculous disease of the glands after measles and whooping-cough may be explained. That these changes in the glands are not necessarily secondary to tuberculous disease of the lungs or pleura is evidenced by the fact that we often find the changes in the lungs recent while those in the glands are obviously of long standing, and if we find in a given case the region of the root of the lung to be the part most affected, it is most probable that the disease originated in the glands and spread outwards. The frequency of caseation of the bronchial glands is shown by the fact that in a series of 300 post-mortems made on children under twelve years of age the writer found the bronchial glands caseous in 110—*i.e.* in 37 per cent of the cases. Such glands may undergo various changes, becoming fibrosed or calcified, or they may undergo softening or suppuration.

Caseation of the glands is usually associated with a tuberculous lesion in the lungs or elsewhere, but may be the sole tuberculous manifestation present. An analogous condition is met with in the abdomen when we find caseous mesenteric glands as the sole tuberculous lesion. The writer believes that caseous bronchial glands are more common than caseous mesenteric glands, thus indicating that tuberculous infection takes place more frequently through the air-passages than the alimentary tract.

Apart from tubercle, metastatic deposits in the glands are of frequent occurrence in association with malignant deposits in the lungs, pleuræ, and œsophagus either of a primary or secondary nature.

Primary new growths in the glands are most commonly of the nature of sarcomata, especially lympho-sarcomata (see "Mediastinal Tumours").

In lymphadenoma (*q.v.*) the bronchial glands take part in the general glandular enlargement.

In both secondary and tertiary syphilis the bronchial glands may share in the general glandular enlargement.

When waxy disease is widespread the glands throughout the body may be the seat of deposits of waxy material, but it is very doubtful if any distinct symptoms or physical signs can be traced to this condition in the bronchial glands.

Disease of the bronchial glands produces effects in two ways—

(i.) By impairment of function.

(ii.) By the production of enlargement with consequent pressure effects.

The effects of impaired function have been briefly referred to above, and are more fully dealt with under the head of tuberculosis. Practically enlargements of the bronchial glands sufficient to cause pressure effects are due to either tubercle or new growths, and the former is infinitely the more common in children, the latter in adults. Although disease of the bronchial glands is not uncommon in adults, it is chiefly in children that definite symptoms are caused, and the subsequent remarks refer chiefly to them.

Pressure effects may be exercised on the following parts:—(i.) The trachea and bronchi; (ii.) the lung; (iii.) the blood-vessels; (iv.) the nerves; (v.) the œsophagus; (vi.) the pericardium.

(i.) *Pressure on the Trachea and Bronchi.*—Some degree of narrowing or displacement is not infrequently seen. In other cases inflammatory adhesions with the wall of the trachea or bronchus develop, and in this way a path is opened up for the infection of the air-passages with tubercle. Perforation of the air-passages is not uncommon. In 800 autopsies on children it was present in 25 cases, *i.e.* over 3 per cent. The right bronchus is more frequently perforated than the left. This may be due to the shorter length of the right bronchus, and to the larger number of glands on that side. The trachea is less frequently perforated than the bronchi. After perforation the softened contents of the glands may be discharged, and the perforation close by cicatrisation, the scar left being small and not tending to cause stenosis of the tube as in cicatrisation of syphilitic lesions. When perforation occurs there is a liability to the occurrence of hæmorrhage from erosion of vessels, but this is rarely so severe as in the cases where the lung itself is involved. Fatal asphyxia may follow the impaction in the glottis of a large caseous mass in process of discharge. Mediastinal abscess with surgical emphysema followed perforation in one case of the writer's, reported in the *Practitioner*, June 1895.

(ii.) Pressure on the lung itself results in inflammatory adhesions, and in this way the lung tissue becomes involved, and infiltrated with caseous material which tends to soften and form cavities. These are not uncommon in the lower lobes of the lung. Malignant disease of the bronchial glands usually extends inwards along the bronchi, compressing them, and frequently producing a bronchiectatic condition of the lung.

(iii.) The effects of pressure on the blood-vessels show themselves chiefly on the veins of the head and neck, which become overfilled, and these parts become congested and even œdematous. The smaller veins may have their walls invaded by tubercle, and thus become the means of producing a generalised tuberculosis. Erosion of branches of the pulmonary artery and vein in the lung may give rise to profuse and even fatal hæmorrhage. The arteries are much less commonly affected, though cases are on record where a cretaceous gland has ulcerated into the aorta and produced fatal hæmorrhage.

(iv.) The effects of pressure on the nerves in the case of caseous bronchial glands are very difficult to distinguish from those due to pressure on the air-passages themselves, but there are, at the same time, cases where pressure on the recurrent laryngeal nerve of one side has produced unilateral paralysis of the vocal cords. The vomiting which occurs in association with enlarged glands, as well as in some cases of pulmonary tuberculosis, has been attributed by some to the effects of pressure on the branches of the vagus nerve. Malignant growths in the bronchial glands may, however, produce marked pressure effects on the nerves (*vide* "Mediastinal Tumour").

(v.) The effects of pressure on the œsophagus are evidenced by some narrowing of that tube and consequent dysphagia. Adhesions may form between the glands and the œsophagus, and if cicatrisation occurs the wall of the œsophagus may be dragged on, and thus a traction diverticulum result. Perforation of the œsophagus by a caseous gland is not of very rare occurrence in children; in examining the records of over 3000 cases in children there were thirteen such cases, or about 0·4 per cent. Perforation of the œsophagus may be accompanied by profuse and even fatal hæmorrhage. When rupture into the air-passages occurs at the same time, gangrenous processes are liable to be set up in the lungs or mediastinum.

(vi.) Pressure on the *pericardium* may cause adhesion between the pericardial sac and the glands, and perforation into the pericardial sac may follow, and then a tuberculous or suppurative pericarditis may be set up. Perforation of the pericardium takes place, most commonly in the upper part of the serous sac.

In addition to these events we may find a combination of two or more of the above-mentioned conditions occurring simultaneously. The writer has seen a case where caseous bronchial glands had perforated the œsophagus, right bronchus, right lung, and the pericardium.

Symptoms and Physical Signs.—Enlarged bronchial glands may exist without giving rise to any symptoms, and to physical signs of very little diagnostic value.

The chief symptoms produced by them are cough, vomiting, dyspnœa, dysphagia, and pain.

Cough.—This is paroxysmal in character, and closely resembles that of whooping-cough. As a rule it is unaccompanied by any expectoration or whoop, but when severe may be accompanied by vomiting.

When the trachea or bronchi are being invaded by caseous glands there is usually some tracheitis and bronchitis, and this is not infrequently accompanied by hæmoptysis varying in amount from a mere streak of blood to profuse or even fatal hæmorrhage.

In some cases the cough has a distinctly clanging character. The voice, as a rule, is not altered, but sometimes the cry has a distinct stridor. The paroxysms of coughing are sometimes so severe that after them the child will fall back quite exhausted. Epistaxis may accompany a fit of coughing, but much less commonly than cyanosis. When a caseous or cretaceous mass ulcerates through the air-passages, in addition to the hæmoptysis mentioned, the caseous or cretaceous mass may be expectorated.

Vomiting is present in some cases, and generally accompanies severe attacks of coughing. When the œsophagus is involved there may be hæmatemesis, and the passage of blood in the stools, or if the air-passages are involved at the same time, food may be expectorated.

The breath in a large number of cases of ulceration into the air-passages has a very offensive smell, though fœtor of the breath does not necessarily indicate ulceration into the œsophagus.

Dyspnœa is sometimes well marked, the patient being unable to lie down in bed, and dreading any movement or interference such as for feeding or changing.

The dyspnœa in some cases is more of the nature of an expiratory than an inspiratory dyspnœa.

When the recurrent laryngeal nerve is involved by a new growth in the bronchial glands the vocal cords may be paralysed (*vide* "Mediastinal Tumour").

Dysphagia may be associated either with pressure of enlarged glands on the œsophagus, or with actual ulceration of a caseous gland through the œsophageal wall. It is not a very common symptom.

Pain when present is generally referred to the region of the manubrium, or less commonly to the epigastrium. When the œsophagus is involved there may be some pain in swallowing, although extensive ulceration of that tube may be present without giving rise to any symptoms.

The physical signs produced by enlarged bronchial glands are often masked by the signs of other concurrent disease in the chest.

Inspection.—The face is frequently cyanosed, especially after an attack of coughing, and there is often marked dyspnœa with recession of the lower part of the thorax: this recession is, as a rule, bilateral in small children, but in older children in whom the thoracic parietes are firmer there may be distinct unilateral recession. The lower part of the sternum is often retracted during inspiration. The veins over the front of the chest are frequently enlarged. A moderate amount of œdema of the face may be present.

Palpation, in addition to confirming the results of inspection, may reveal the presence of enlarged glands in the neck or axillæ. The presence of surgical emphysema may also be verified. Vocal fremitus, or in small children the cry fremitus, may be diminished over the area supplied by a compressed bronchus, or increased over the manubrium.

Percussion.—Impairment of resonance of the percussion note may be present over the manubrium and inner end of the first or second intercostal spaces anteriorly, and similarly in the suprascapular fossæ and interscapular region posteriorly. The percussion of the back is much less reliable in small children than in adults. Special care should be taken in examining small children to see that the child sits up or is placed quite straight, and not allowed to lie over to one side, as it is so apt to do when being held by the mother or nurse.

Auscultation.—The breath sounds over the lungs may be simply weak, while over the dull area there is often marked tubular or bronchial breathing, and in some cases there may be almost amphoric breathing. Vocal resonance over the dull area is increased, and may have an œgophònic character. When catarrh of the air-passages exists there are the usual signs of this condition, and should these be unilateral in distribution, suspicion should be directed to the bronchial glands. The usual sequence of auscultatory signs in the lungs is—weak breathing followed by bronchial breathing, to which fine crackling râles, and eventually sonorous and bubbling sounds, are added.

Where the lung is involved either in tuberculous processes or by the formation of breaking-down areas, these signs become masked by those of pulmonary tuberculosis, or empyema, or the signs of bronchiectasis.

Eustace Smith has pointed out that a purring sound can be produced in the vessels of the neck during respiration by making the child look upwards, and so extend the neck and compress the vessels by the retromanubrial glands. The stethoscope must be applied below the suprasternal notch.

If signs of tuberculous consolidation are present and are limited to the middle lobe, this would suggest the bronchial glands as the *fons et origo mali.*

DIAGNOSIS.—It must be borne in mind that the condition frequently exists without giving rise to any symptoms; and further, that the physical signs are often equivocal. Every condition liable to produce paroxysmal cough and dyspnœa must be differentiated. Of these the more important are whooping-cough, enlarged tonsils, pulmonary tuberculosis, empyema, and bronchiectasis.

In other cases asthma, a foreign body in the air-passages, adherent pleura, pressure on the air-passages by aneurysms, abscess, a new growth, prevertebral abscess in the upper dorsal region, laryngeal diphtheria, and laryngismus stridulus must be excluded. Careful consideration of the history and detailed examination of the pharynx, larynx, and chest will usually suffice to differentiate these conditions.

PROGNOSIS.—When the enlargement is due to malignant disease this is necessarily hopeless, but when due to tuberculous disease it is not of necessity so grave, and yet it must be borne in mind that tuberculous meningitis not infrequently is met with in cases in which the bronchial glands are the only recognisable sites of previous tuberculous infection. Recovery is possible even when the air-passages or the œsophagus are perforated.

TREATMENT.—The treatment of enlarged bronchial glands is that of the diseases which cause the enlargement.

Prophylactic treatment is of great importance. After an attack of measles, whooping-cough, or influenza, when all risk of infection is past, a stay at the seaside, in a part having a sheltered and southern aspect, is advisable, or else in some high and dry locality sheltered from the east wind. Clay soils are to be avoided. The clothing should be warm, woollen underclothing being best, and the limbs should be entirely covered up. It is astonishing how much thoughtlessness is displayed in the exposure of the lower limbs of children to the air out of doors, both in health and after illness, in the erroneous belief that it is "strengthening" to them!

Special precautions should be taken with the food, all milk being boiled, unless the absence of tuberculous disease in the cows has been ascertained by inoculation.

The dwelling and sleeping rooms should be large, light, well ventilated, free from dust, and the windows should be kept open at night.

The medicinal treatment of these cases consists in the administration of some form of cod liver oil in combination with iron, or maltine, or the hypophosphites.

The syrup of the iodide of iron, in doses of 5-10 minims three times a day, often produces marked diminution of enlarged glands in the neck, and has proved useful in cases where the bronchial glands were also enlarged. Sulphide of calcium has been recommended in doses of one-twentieth to one-tenth of a grain every hour. Arsenic may be given in combination with iron, or with a vegetable bitter. Iodine in the form of the tincture, in doses of one to three minims, may be tried.

For the treatment of the cough and vomiting the writer advocates the employment of the glycerine of carbolic acid, in doses of one to three minims, in combination with cod liver oil and iron. Creasote in capsules is also useful.

If complications arise, *e.g.* tuberculosis of the air-passages, erosion of the œsophagus, empyema, etc., they should be treated along the lines indicated under these headings.

Tracheotomy is only of use when a caseous mass is impacted in the trachea or glottis.

LITERATURE.—1. QUAIN'S *Dictionary of Medicine*, vol. i.—2. VOELCKER. *Practitioner*, June 1895.—3. *Transactions of the Pathological Society of London*, 1898.

Bronchitis.

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BRONCHITIS is an inflammation of the bronchial tubes, general or partial, involving either the larger or smaller bronchial tubes.

The term bronchitis is of comparatively recent date, having been introduced by Dr. Charles Badham in 1810, when it superseded the old phrase "pulmonary catarrh" or "defluxion."

For the purpose of description the subject will be divided into the following sections:—1. Acute bronchitis in the adult; 2. Chronic bronchitis in the adult; 3. Bronchitis in children.

General Considerations.—It would naturally be supposed that the bronchial tubes, ramifying throughout the whole of the lungs as they do, should be so intrinsically a part of the lung that no clinical division could be drawn between inflammation of the bronchi and inflammation of the pulmonary tissue, pneumonia. And though from a clinical as well as from a pathological standpoint bronchitis is sharply differentiated from pneumonia so long as the larger and middle-sized tubes are alone affected, we shall seek in vain for any line of demarcation between capillary bronchitis and the lobular pneumonia with which, in children at any rate, it is almost invariably associated.

To appreciate the pathological changes taking place in bronchitis, it is necessary to bear in mind the normal structure and functions of the tissues involved. An explanation will be found for the tendency for the inflammation to be mainly bronchial, firstly in the anatomical arrangement of the blood-supply, and secondly in the protecting action of the basement membrane, while even the marked variation in the clinical characters between bronchitis affecting the larger and smaller tubes very largely depends on their different anatomical structure and relations. The noteworthy point in regard to the vascular system of the lung consists in its double circulation, the blood-supply for nutrition and function being separately provided for; thus the main bronchi and the tissues of the lung are supplied, not by the pulmonary artery, but by the bronchial arteries which are derived from the aorta. Furthermore, the bronchial veins communicate freely with the pulmonary artery, and in the smaller bronchi the blood is returned almost wholly by branches of the pulmonary vein to the *left* side of the heart: consequently any obstruction to the return of blood to the left side of the heart will at once result in congestion of the *smaller* bronchi. Moreover, while it seems probable that the pulmonary arteries are not subject like most arteries to vasomotor control, this does not apply to the bronchial arteries, and thus a variety of causes acting either directly, or indirectly by reflex action, are able to cause a vascular dilatation which may be limited to the bronchial tubes, as distinguished from the air-vesicles which are supplied by the capillary plexus of the pulmonary vessels, and which are directly concerned with the function of respiration—aeration of the blood.

The right and left bronchi divide and subdivide dichotomously within the lung until ultimately the minute terminal bronchioles end in the infundibula and air-vesicles. In a transverse section of one of the middle-sized bronchi three coats may be distinguished—the inner mucosa, the middle muscularis, and the outer fibrous coat, which contains the cartilages. Further examination shows that the mucosa is composed of (1) an epithelial lining with three layers of cells—the single layer of superficial columnar ciliated epithelium, an intermediary layer of transitional or rounded cells, actively germinating to form the superficial columnar cells and mucous corpuscles, and a single deep layer of flat nucleated cells; (2) an inner fibrous coat, abundantly supplied with branches from the bronchial artery, and consisting of bundles of fibrous tissue with numerous lymphatic spaces containing lymphatic corpuscles between them; and (3) a homogeneous, hyaline, structureless basement membrane giving attachment to the epithelial covering which it effectually separates from the vessels and lymphatic vessels of the inner fibrous coat. Hamilton has shown that the part played by this basement membrane in bronchitis is of great importance, in that it imparts a superficial character to the catarrhal affections of the bronchi; for though it becomes swollen and œdematous in bronchitis, and permits the transudation of serous fluid into the lumen of the tube, no leucocytes appear to pass from the deeper structures to the surface of the mucous membrane, nor can inhaled particles of foreign matter, like coal-dust in a miner's lung, become carried down to the tissues of the bronchi. Such particles do not enter the lymphatics of the lung except through the air-vesicles. In the middle-sized and smaller bronchi the muscular coat is well developed, and it is continued down to the infundibula, where it is represented only by a few scattered fibres. The physiological action of this muscle is not known exactly, but it probably regulates the intra-alveolar air-tension, and protects the air-vesicles from undue pressure in coughing and other respiratory acts. Consequently, as Hamilton has demonstrated, this coat generally becomes greatly hypertrophied in chronic bronchitis, owing to the constantly recurring cough. Gairdner, in discussing the action of expectorant medicines, arrives at the conclusion that expectoration depends more upon the expulsive mechanism of these muscular fibres than upon the alterations in the consistency of the secretions. Their action is probably somewhat analogous to intestinal peristalsis, and the beneficial result of emesis in certain cases of bronchitis is due to the remedies increasing the action of the bronchial muscles directly, rather than to the vomiting, which is perhaps only a concomitant, but in no way essential, effect. Further evidence of this important physiological action is afforded by the fact that, when the muscular coat loses its functional activity, as in the more serious cases of bronchitis, the bronchial tubes dilate and become filled with secretion. Walshe, referring to Radcliffe Hall's researches, considers that in the calm, and still more in the forced respiration of health, the muscular coat possesses the power of reducing the tubes to less than their medium size. But in bronchitis the muscular coat loses its contractile force, the elasticity of the inflamed bronchi being likewise impaired, and so becomes intelligible, the tendency to dilatation of the tubes, the laboured expiration and the accumulation of mucus.

It is not improbable that, like the alæ nasi and vocal cords, the bronchial muscle may rhythmically dilate and contract with inspiration and expiration. I believe that this largely explains why in bronchitis and asthma the dyspnœa is expiratory rather than inspiratory, inasmuch as the muscular spasm is more or less inhibited during inspiration, permitting air to be inspired more freely than during the period of bronchial spasm it can be expired, consequently the lungs become distended and emphysematous. In the larger and middle-sized bronchi the outer fibrous coat contains cartilaginous plates, but not in the smallest bronchi, which consist of a layer of stratified ciliated epithelium, surrounded by the muscularis and peribronchial connective tissue. If one of the bronchioles be followed towards its termination, it is found that just before it ends in the infundibulum the columnar ciliated epithelium is replaced by a single layer of cubical epithelium, a thin fibrous tissue investment being still present, and finally when the air-vesicle is reached this epithelial layer comes to be composed of a very thin layer of flat, squamous, endothelial-like cells, while the muscular coat is lost.

The thinness and weakness of the walls of the smallest bronchi, and the feeble expulsive power of their muscular coat, together with the narrowness of the lumen of the tube, and especially the complete absence of ciliated epithelium and of any muscular coat as they approach the infundibula, explain the serious nature of a capillary bronchitis and the danger resulting from secretions accumulating in the smallest tubes.

I. Acute Bronchitis in Adults

Etiology.—We may divide the causes of acute bronchitis as follows:—

(*a*) *Predisposing.*—No age is exempt, but it is especially liable to attack the very old or the very young, and to assume a serious form in these subjects. Males and females are equally liable to suffer. Sedentary life and luxurious habits, and living much indoors and in vitiated atmosphere, heart-disease, especially those forms which result in pulmonary congestion, and gout, diabetes, rickets, dyspepsia and constipation, spinal curvature, dusty occupations, and a cold and changeable climate, all predispose to bronchitis. But heredity and a certain individual susceptibility to catarrhal affections undoubtedly largely influence the occurrence of attacks, and, above all, the pre-existence of various pulmonary affections and the fact of previous attacks of bronchitis having taken place.

(*b*) *Exciting causes.*

1. Exposure to cold; "catching cold."
2. Traumatic; the inhalation of steam; swallowing hot water or irritating fluids.
3. Chemical irritants, *e.g.* the inhalation of irritating gases or vapours such as chlorine or ammonia, sulphurous or nitrous acid, ether, iodine, bromine, etc.
4. Toxæmia, *e.g.* in Bright's disease, gout, rheumatism.
5. Vasomotor, *e.g.* asthma.
6. Infective, *e.g.* typhoid, typhus, measles, scarlatina, small-pox, malaria, whooping-cough.

Acute bronchitis is generally associated with exposure to cold, and it is certainly influenced by climatic conditions. But although a so-called chill is undoubtedly a very common exciting cause of bronchitis, it is equally certain that in many cases the real cause of the attack is by no means so simple. Indeed we find that it is not those whose daily life constantly exposes them to cold, or even rapid changes of temperature, that are most prone to attacks of bronchitis, "persons who live an out-of-door life are usually less subject to the disease than those who follow sedentary occupation" (Osler). Certain it is that colds are not usually caught in pure air, at sea for instance, or in the higher Alps in winter, although exposure to cold is of necessity frequent. Acute inflammatory attacks of the upper air-passages are most frequently found in those who habitually breathe vitiated air, or who have attended over-crowded, ill-ventilated places of meeting, and are then exposed to cold, or who are directly infected by some person suffering from a cold in the head. Not only do these "catarrhs" confer temporary immunity, but recent investigations, such as those of W. H. Park, have shown that staphylococci and streptococci, which are always present in the healthy mouth, increase greatly in number and virulence in damp weather and in winter months, and are then capable of setting up acute pharyngitis if applied to the throat. These are just the conditions which are particularly prone to lead to bronchitis, and we thus seem to have an explanation of its frequent occurrence in the changeable weather of early spring and late autumn, and of its origin in a "cold on the chest."

Sputum normally contains a considerable number of different varieties of micro-organisms, some of which are best studied on stained films, whilst others grow well on the ordinary media. Amongst the most common of these organisms are the streptococcus brevis and other varieties, staphylococcus albus, citreus, and aureus, spirillum sputugenum, leptothrix, and varieties of chromogenic bacteria, bacilli and sarcinæ. Whilst any one of these may be found in the sputa of bronchitis and broncho-pneumonia, it will commonly be found that one or more organisms are present in overwhelming majority, so that prepared films or cultures may show almost a pure culture of one particular variety. Pansini found in normal air-passages several different streptococci, bacilli, micrococci, sarcinæ. Queyrat describes a specific organism, a micrococcus of "tracheo-bronchitis." Grun describes a specific bacillus of bronchitis, but neither of these observations has been substantiated. Netter gives the pneumococcus as the cause of capillary bronchitis and broncho-pneumonia in 15·85 per cent of cases.

Osler makes the statement that acute bronchitis is probably a microbic affection, and Gee, in referring to the effect of dust in certain occupations in causing bronchitis, states that the dust of unclean and ill-ventilated rooms will be very likely to contain morbific microbes, and that the irritation of the air-passages set up by inanimated dust will favour the operations of the many bacteria which are potent to cause bronchitis. He believes that our mouths, noses, and throats harbour many morbific microbes in a latent state, there awaiting a favourable opportunity for becoming active and virulent, and thus setting up the catarrhal attack which spreads down to the trachea and bronchi.

No doubt many of the cases of tracheo-bronchitis originate in this manner; yet, notwithstanding the bacteriological researches which have been just mentioned, I am convinced that in a larger proportion of the cases of bronchitis in which the middle-sized bronchi are mainly implicated from the outset, exposure to cold is itself the immediate cause of the disease. It may be brought about directly by cold air entering through the upper air-passages, as shown, for instance, by the experience of susceptible persons on going from a heated atmosphere into cold night air, by the liability to bronchitis in tracheotomised patients, or in habitual mouth-breathers owing to the inspired air not being duly warmed by passing through the nose. But in most cases it seems that the action of cold is indirect, and the result of vasomotor paralysis, as explained by Rosenthal; for often enough the cause of the attack has been getting wet, or lying on damp grass, or sitting still after getting over-heated and perspiring, and not by the action of cold air entering through the mouth.

Other deep-seated viscera having no air-containing tubes of communication to the surface of the body, such as the bladder and kidney, are similarly and specially subject to inflammatory attacks from exposure to wet and cold applied to other parts of the body, and in applying hot fomentations or poultices to the chest, to the hypogastrium or lumbar regions in inflammations affecting respectively the lungs, bladder, or kidneys, we take advantage of these associated reflex vasomotor areas.

We may further refer to the secondary bronchitis which is liable to arise in the course of influenza, typhoid fever, measles, and other exanthemata. It is widely recognised that the bronchitis of influenza, which is certainly microbial, is especially liable to be aggravated by the least exposure to cold, while the pulmonary complications of measles are particularly prone to be severe if the rooms are at all crowded or ill-ventilated. It is a remarkable fact that influenza epidemics are just as prevalent at Davos and St. Moritz as in the less dry atmosphere of England and Scotland, and that, despite the ideal climate of the higher Alps for most bronchitic affections, cases of influenza at these Swiss resorts are just as prone to be complicated with severe bronchitis and pneumonia. Thus a chill, while undoubtedly capable of directly causing acute bronchitis in those unaccustomed to exposure, or in those who seem to have a special liability to catarrhal affections of the respiratory tract, is often an indirect rather than a direct influence in causing the disease, and there are the other causes of bronchitis which can in no way be associated with either cold or micro-organisms, such as inhalation of irritant vapours, such as strong ammonia, chlorine gas, etc.

Morbid Anatomy.—The morbid changes underlying bronchitis in its various forms are primarily an exaggeration of the normal physiological processes taking place in a healthy lung, and we shall find that inflammation of the bronchial tubes is essentially similar to inflammation in other mucous membranes, with certain modifications due to their peculiar anatomical structure and relations, so that the course of the disease, and the particular clinical type it assumes in different cases, vary not only with the actual cause or the acuteness of the attack, but depend in a large measure on the calibre of the bronchial tubes most concerned.

It is not very often that we are afforded an opportunity of examining post-mortem the actual conditions presented by any but the directly fatal and therefore most severe forms of bronchitis. Socoleff, who induced bronchitis in animals by the application of chemical irritants, and Hamilton, from post-mortem observations on patients dying with bronchitis, have investigated the histological changes which occur in the bronchi in health and disease, and I am mainly indebted to the writings of the latter for the following description of the morbid anatomy of bronchitis.

The initial change is hyperæmia, followed by swelling of the mucous membrane and excessive secretion. The hyperæmia, as Biermer demonstrated, may first affect either the superficial or deeper structures, or both.

According to Hamilton, the earliest departure from the normal condition is a relaxation and distension of the attendant plexus of blood-vessels ramifying in the inner fibrous coat, immediately beneath the basement membrane—that is to say, of the branches of the bronchial artery. In a few hours the basement membrane became swollen from serous infiltration. Next, and quite early in the course of the affection, Socoleff and Hamilton found that the ciliated epithelial layer desquamates, and is not regenerated till recovery takes place, when it is gradually reproduced. The cells of this layer undergo fatty degeneration, and Hamilton says that it is no doubt partly destroyed by this means and partly expectorated, while others may be inhaled into the smaller bronchi, where they may be seen lying in large detached masses among the other catarrhal products. Wilson Fox and Biermer consider that the epithelial desquamation is largely a post-mortem change, and Hamilton admits that the shedding is seldom complete, the deeper layer of transitional cells, or at least the single layer of flat germinating cells, usually remaining, and by a process of fissiparous diversion rapidly reproduces the nucleated cells, which, being continually thrown off, give to the secretion its peculiar catarrhal character. At the same time the mucous glands are particularly active, and it is the combination of this catarrhal mucus with the epithelial cells which gives rise to the so-called muco-purulent fluid which occupies the bronchial lumen until removed by expectoration. After about the tenth day, Hamilton found that the congestion not only affects the inner fibrous coat, but that all parts of the bronchus exhibit vessels over-distended and engorged with blood, and that, *pari passu* with the changes in the epithelial covering, the inner fibrous coat becomes infiltrated with cells derived from the endothelium lining the lymphatic spaces, until the whole of the lymph-spaces become choked by these new cellular products. These cells, according to Hamilton, never extend towards the free surface of the mucous membrane, the basement membrane forming an impassable barrier in that direction, but they invariably spread outwards, through the inter-muscular lymphatic spaces, to the adventitia, where similar changes will be found to have taken place. But this proliferation, with the production of leucocytes, soon spreads throughout the lobular septa to the deeper layers of the pleura, the whole of the lymphatic vessels becoming the subject of a catarrh, while the lymphatic glands at the root of the lung are then invariably found enlarged.

The hyperæmia of the mucous membrane may almost entirely disappear after death, but as a rule it is bright red, or in cases of longer duration dark purple, and presents a thickened, opaque, and velvety appearance, the lumen of the smaller tubes being diminished by these changes, and filled with thick, opaque, yellow secretion which exudes from the small bronchi from the cut surface of the lung on pressure. Bronchitis of the smaller tubes is accompanied by pulmonary congestion and œdema, and generally areas of collapsed lung or foci of lobular pneumonia may be found.

Again, Hamilton has found that the first indication of recovery taking place in bronchitis seems to be the diminution of the congestion of the mucous membrane, the vessels apparently recovering their tone, the proliferation of the epithelium becoming less active, the cells being once more fully developed into columnar cells, while the cellular infiltration of the bronchial wall and the lymphatic vessels becomes gradually absorbed in cases which proceed to recovery.

In those cases which from the severity of the attack, or from failure of the factors which make for recovery, or which from the outset, owing to the persistence of less acute causes, pass into the condition of chronic bronchitis, the mucous membrane and entire bronchial wall remain thickened, the muscular coat becomes hypertrophied, while the mucous glands and the bronchial cartilages more or less completely disappear, being replaced by dense cellular infiltration. In advanced cases, however, the muscularis and the adventitia become atrophied, probably owing to the cellular infiltration of the lymphatics, and thus arise those changes which from various causes result in bronchiectasis, emphysema, interstitial pneumonia, and other pulmonary complications which are beyond the scope of this article.

Collapse of the Lung.—A collection of the catarrhal secretion of bronchitis may temporarily occlude a smaller bronchus, resulting in weakness or absence of the vesicular murmur over the corresponding portion of the lung. On the patient coughing, the occluding mass of secretion may be dislodged, the air again entering the air-vessels. But the inflammation may extend to the smallest bronchioles, or the muco-purulent secretion may be sucked down so as to plug a bronchial channel which itself is unaffected. Especially if in any part of the lung several of these finer divisions are occluded, no effort of coughing may suffice to dislodge it. The air in the corresponding portion of the lung then becomes absorbed by the surrounding pulmonary capillaries, and the air-vesicles collapse, with more or less so-called "compensatory emphysema" in the other portion of the lung. Collapsed lung has a pinkish red appearance, and on section is reddish brown, giving a frog-spawn sensation to the touch, and the corresponding surface of the lung is depressed. Merely collapsed lung does not as a rule become inflamed, unless it be from extension of inflammation from a neighbouring patch of pneumonia, and on the removal of the obstructing cause, if this be not too long delayed, may again become inflated. But coincidently with the occurrence of collapse, patches of lobular pneumonia, with true red hepatisation, are usually found, probably due either to extension of the inflammatory process right down to the air-vesicles and pulmonary stroma, or to direct infection by various micro-organisms. (For fuller details see "Pneumonia.")

SYMPTOMS.—Acute bronchitis in its clinical aspects presents every gradation from a mild and transient cold on the chest to an intensely acute and rapidly fatal disease attended with urgent dyspnœa, cyanosis, and collapse. In the milder forms, or tracheo-bronchitis, the attack is usually ushered in by the symptoms of an ordinary "cold," with lachrymation, sneezing, and a stiffness and soreness of the throat. The catarrh may extend gradually to the chest, involving the larynx with resulting hoarseness, and causing a sense of rawness and soreness behind the sternum. In other cases the cold seems to settle on the chest from the outset without obvious pharyngitis and laryngitis. Initial rigors are not usual; but a sense of chilliness, shiverings, pains in the bones and back, general malaise, headache, drowsiness, and languor, with more or less gastric catarrh, hepatic disturbances, and constipation, commonly characterise the earlier stages. The pulse and respiration are moderately quickened; the skin, at first dry, soon becomes moist.

In still other cases, particularly in the old or very young, the onset may be almost sudden, with severe cough and urgent dyspnœa, as Wilson Fox describes it, almost reproducing the phenomena of spasmodic asthma, but differing from the latter in its persistence; or cough and dyspnœa may set in acutely, but without so distinct spasmodic element, followed after a few hours by the expectoration of an abundant, glairy, blood-stained mucus.

In milder cases the temperature is but slightly raised; but with the more severe it may range between 101° F. and 102° or 103°, showing the usual evening rise and the morning decline.

The urine is of the usual febrile character, of high specific gravity, high coloured, loaded with lithates and urea, and free from albumen unless complications are present.

The cough at first is harsh, loud, and ringing, either coming in frequently repeated single coughs, or in paroxysms which are exceedingly harassing to the patient, accompanied by a sense of oppression and tightness in the chest and a considerable soreness beneath the sternum and along the attachments of the diaphragm, the severe paroxysms sometimes ending in vomiting. It is often most distressing in the earliest stages before there is any expectoration, being then due to irritation of the inflamed bronchial mucous membrane, the irritation being referred to the laryngo-tracheal region. After an interval the cough is attended with expectoration, which at first is thin and watery, and frothy, consisting of serous exudation containing the ciliated epithelium which is shed very early. As the disease progresses, it becomes more consistent and ropy, from the increased exudation of mucus, and more or less opaque, owing to the numerous round cells thrown off from the deeper epithelial layers, becoming in turn muco-purulent and purulent. The cough is then due, not to irritation of the dry mucosa, but to the accumulating secretions which require to be got rid of, and is therefore apt to be more severe on rising from the recumbent position after sleep. Streaks of blood are sometimes seen in the sputum, especially in the earlier stages when the cough is severe.

Dyspnœa is seldom pronounced when the larger tubes alone are affected. When the smaller tubes are extensively implicated, owing to the excessive contraction of the muscular coat acting in the manner explained above, and perhaps in part from the valve-like action of the accumulating secretion, more air is drawn into the lung than can be expired, and the lungs consequently become over-distended, and the respiratory movements shallow and ineffectual. It is remarkable that the dyspnœa is abdominal and expiratory, rather than inspiratory, in contradistinction to the dyspnœa due to obstruction in the larynx or trachea. When the air-vesicles are already distended with the air which no effort of the patient can effectually expire, but little or no more air can enter, and thus there is not much tendency for the secretion to be sucked in. Expectoration is then attended with much difficulty, and the thick, yellow, sticky secretion from the small tubes is seen to hang in strings suspended in the lower watery layer from the upper layer of frothy mucus from the upper larger bronchi. Sometimes the expectoration is very scanty in capillary bronchitis, even when moist râles are audible over the chest.

An attack of acute bronchitis may assume an asphyxiating type, either from a simple bronchitis of the larger tubes occurring in a debilitated patient, especially in the emphysematous, or from a very extensive implication of the smaller bronchi, or from widespread involvement of the capillary bronchi. Though capillary bronchitis in the adult may be remarkably sudden and severe in its onset, it not infrequently commences with the symptoms of an ordinary mild bronchitic attack. Rigors are rare, says Walshe, and vomiting rarer. But nervous symptoms supervene early and are pronounced, the patient is restless and drowsy, the pulse rapid and small, cyanosis is a marked feature, and there is complete loss of appetite. Muttering delirium at night and sleeplessness is common, but acute maniacal excitement is not unknown. The acute dyspnœa, cyanosis, and active, sometimes maniacal, delirium when strong, young adults are the subject of acute capillary bronchitis, form a very painful clinical picture. If the attack cannot be relieved the patient rapidly becomes asphyxiated. Walshe, in his description of this type of bronchitis, states that, "as long as the strength permits, the patient sits or bends forwards; but the body gradually yields, and it is not uncommon to find patients, while still perfectly conscious, lying sideways or forwards, with the head lower than the shoulders, and in rare cases this posture of the head is adopted from the very outset."

Physical Signs.—The chest may not be altered in shape in mild cases, but in pronounced attacks assumes the inspiratory type; it remains more or less fully expanded, the respiratory movements being elevatory rather than attended with expansion and contraction of the parietes, and are violent, and increased in frequency, though not in amplitude. The abdominal movements are increased, and if the bronchial tubes are extensively involved, there will be marked cyanosis and fulness of the large veins of the neck. The right side of the heart dilates, giving rise to epigastric pulsation; or the heart may be pushed down, the impulse being felt in the left costal angle.

The liver is often congested and displaced downward. But in the acute asphyxiating type, as has already been stated, the respiratory movements of the over-distended chest are increasingly shallow, and the heart's action is apt to fail rapidly, with marked cyanosis, clammy perspiration, coldness of the skin, general anasarca, and scanty albuminous urine.

The adventitious sounds which arise in bronchitis are of two kinds—the dry (rhonchus and sibilus) and the moist (râles). Rhonchus, or sonorous rhonchus, is a loud, deep-toned, cooing sound, due to air passing through one of the larger tubes which is partly obstructed by a collection of mucus, producing a *veine fluide.* The mucus can often be removed by the patient coughing, hence rhonchus audible at one moment may abruptly disappear. Sibilant rhonchi, or sibilus, is a more high-pitched whistling sound, due to swelling of the mucosa in the small bronchi; hence they are of graver importance, and as they cannot be dispelled by coughing, they tend to persist in the same region for a considerable period.

A rhythmic rhonchus or sibilus, as was first observed by Stokes, may be produced by the ventricular contractions, if the portion of lung is in close proximity to the heart and the secretions abundant.

Râles may be large, medium, or small, according to the size of the bronchial tube which is occluded by an accumulation of mucus, the sound being produced by the air passing through the mucus. Moderately fine râles, however, may be produced in the larger bronchi. Large râles are of less grave import than the small, in that they imply that the larger tubes are mainly affected; and mere loudness of the sounds is not of bad omen—in fact, it is in the graver cases of widespread implication of the finer tubes that the moist sounds are least audible. The smaller râles are sometimes spoken of as sub-crepitant. Yet it is of considerable importance that they should be distinguished from the crepitation, which is indicative of pneumonia or of phthisis, conditions which may be associated with the symptoms of simple bronchitis.

A plug of mucus in a bronchus may temporarily prevent the air entering the corresponding portion of the lung, and causes a localised diminution or absence of breath-sounds, until by coughing the plug is removed. But usually in bronchitis the amount of air entering the vesicles being deficient, the normal vesicular murmur is less distinct than in health, consequently the bronchial breath-sounds are less marked and the breathing seems unduly harsh. Yet the bronchial breathing is never heard in uncomplicated bronchitis, except over the roots of lungs; it is only heard when either collapse or pneumonia is present, and may then be associated with dulness on percussion and increased vocal resonance.

Vocal fremitus and voice-conduction are scarcely altered in uncomplicated bronchitis.

The percussion-note is normal unless the bronchitis is pronounced, and then the whole of the upper part of the chest, being acutely distended and emphysematous, yields a hyper-resonant or almost tympanitic note. Even small patches of broncho-pneumonia do not give rise to dulness on percussion, but congestion of the bases, collapse, or pneumonia, if extensive, causes a diminution of the resonance. The tongue is almost invariably coated, and symptoms of gastro-intestinal catarrh, with anorexia, thirst, and constipation, are generally present.

In uncomplicated cases of the larger and middle-sized tubes the attack gradually subsides in the course of a week, the expectoration becoming decreased in amount and more normal in character, the physical signs in the chest gradually disappearing, convalescence being established in about ten days or a fortnight. But in the old and feeble, and in young children, the disease is apt to extend down to the smaller tubes, with a liability to pulmonary collapse. Especially is this dangerous complication likely to arise in measles and whooping-cough. In the acute suffocative type the prognosis is always grave, even in cases uncomplicated with cardiac or renal disease, while any co-existent affection of the lungs, heart, or kidney greatly aggravates the danger.

Walshe said he had known a case fatal in forty-six hours; but while these patients often succumb within a few days, they sometimes recover even after the condition appears hopeless.

Acute bronchitis may pass into a subacute or chronic state, and is then liable to be associated with various complications.

DIAGNOSIS.—The diagnosis of acute bronchitis rarely presents any difficulty. Though it may be abrupt in onset, and even attended with considerable fever, there is not that initial rigor and characteristic disturbance in ratio between respiration and pulse-rate of pneumonia; while the absence of fine crepitation or tubular breathing, and the normal and exaggerated resonance of the chest, the widespread presence of coarse râles and rhonchi are sufficient to differentiate bronchitis from pneumonia.

Acute pulmonary tuberculosis may closely simulate, and for a time be indistinguishable from, acute bronchitis. High pyrexia and great prostration with early delirium, while not excluding simple bronchitis, suggest acute tuberculosis; the occurrence of hæmorrhages may assist the diagnosis in doubtful cases (Wilson Fox).

It is, however, necessary to bear in mind that bronchitis may be pronounced in the early stages of typhoid fever, measles, whooping-cough, and more rarely variola, scarlatina, and other affections, the indications of which must not be overlooked. Thus the initial lacrymation, conjunctival injection, sneezing, and rhinorrhœa would lead to the suspicion of measles. Headache, general malaise, the peculiar rise of temperature, tenderness and gurgling in the right iliac region, enlargement of the spleen, the characteristic rose-spots and other indications of typhoid fever should be eliminated. The aching in the lumbar region in variola and the sore throat of scarlatina and early appearance of the rash seldom leave one in doubt for any considerable period. Whooping-cough may closely simulate simple acute bronchitis, and prior to the development of the characteristic crowing, may be indistinguishable.

TREATMENT.—In the milder form of bronchitis of the upper bronchial tract—tracheo-bronchitis—domestic remedies for a cold on the chest usually suffice, and these only call for mention. The mustard and hot-water foot-bath, the application to the chest of a mustard poultice, or hot fomentations sprinkled with turpentine, followed by a Dover powder, and an aperient, and some simple diaphoretic mixture such as sweet spirits of nitre, and small doses of ipecacuanha, will often prove sufficient to relieve and shorten the attack. If the patient cannot remain indoors for a day or two, it is necessary to avoid diaphoretic remedies, which render him more susceptible to cold, and involve a risk of converting a slight bronchial attack into the much graver general bronchitis of the smaller tubes, and for this reason a Turkish bath in the initial stages of bronchitis is attended with a new risk.

In severer attacks more active measures are needful; and it should be ascertained whether the bronchitis is a primary affection, or secondary to some constitutional condition or other disease, *e.g.* Bright's disease, which would call for appropriate measures.

In acute primary bronchitis the patient should be confined to his bed, and the temperature of the room maintained at about 62° to 65° F. We have seen that the first stage is due to vascular enervation and engorgement of the mucous membrane. Thus the chief indication is to combat the dry and congested condition by improving vascular tone and inducing freer secretion.

At the outset a large mustard and linseed-meal poultice should be applied to the front and back of the chest, this being replaced by simple linseed poultices after the mustard has sufficiently stimulated the skin to produce thorough redness. A steam-inhaler, with compound tincture of benzoin or tincture of belladonna added to the hot water, often affords relief. A moist atmosphere is not only comforting to the patient, but seems to materially relieve the dry, hacking cough; but when a steam-kettle is used, it must be remembered that the steam may condense on the bedclothes, and special care is needful by ensuring the maintenance of the temperature of the apartment, to obviate the risk of a chilling effect on the patient. During the early period in the attack the drugs that are most relied on are tartarised antimony and ipecacuanha, apomorphia, or squills, to promote bronchial secretion, together with citrate of potash, or acetate of ammonia, spirits of nitre, or some other diaphoretic. If there is bronchial spasm, tincture of lobelia or belladonna, citrate of caffein, chloral, or opium may be added, but should be cautiously used, especially in children and in the old or feeble.

As soon as the dry stage passes off and secretion commences, antimony and the diaphoretic remedies may be discontinued, and stimulant expectorants, such as carbonate of ammonia with senega, squills, euphorbia pilulifera, or small doses of iodide of potassium, substituted. At this stage the preparations of opium and direct sedatives to check the cough which is due to the accumulation of secretion in the tubes should be avoided.

When the smaller tubes are extensively involved and the amount of secretion is very considerable, the patient must be carefully watched for any indication of cardiac failure or pulmonary collapse. In weakly patients with impaired heart-action it may be necessary to resort at once to alcoholic and other stimulants, such as digitalis, strychnine, or ether.

If the bronchial secretions tend to collect in the smaller tubes and pulmonary collapse is threatening, it may be desirable to induce vomiting by the administration of sulphate of zinc or by repeated large doses of ipecacuanha. Depressing emetics should be avoided.

With threatening apnœa, the use of oxygen inhalations, or, on rare occasions, venesection, may be indicated. It is hardly necessary here to give directions in regard to diet in bronchitis, beyond emphasising the importance of keeping the patient on low diet in the beginning of an attack, for the gastro-intestinal tract is always more or less deranged, and calls for appropriate treatment.

II. CHRONIC BRONCHITIS IN ADULTS

ETIOLOGY.—Chronic bronchitis is due to much the same, though generally more persistent causes, as the acute form; but the influence of predisposing factors and constitutional conditions is more potent, and is more usually the essential cause of the chronic affection. Moreover, chronic bronchitis is distinguished rather by its protracted course and secondary complications than by any special clinical features. In some cases frequent subacute attacks by increasing the susceptibility of the individual, and resulting in a gradual permanent impairment of the tissues, pass insensibly and without any line of demarcation into the chronic form. Thus chronic bronchitis may follow repeated attacks of acute bronchitis, or may be chronic from the beginning. It is usually met with in the aged, the "winter cough" of old people, recurring from year to year throughout the colder months. Various diatheses, such as syphilis, gout, and renal disease, heart affections, especially disease of the mitral valve, aneurysm, and chronic lung affections, such as phthisis, emphysema, and dilated bronchi, and almost any condition impairing general health, predispose to chronic bronchitis. But a mild form is also met with in children and young adults, who suffer periodically from recurrent catarrhal attacks either on the slightest exposure, or it may be associated with gastro-intestinal disturbance. Chronic bronchitis is also very common in workers in dusty occupations, millpuff upholsterers, bakers, colliers, etc., and is often associated with chronic alcoholism.

Morbid Anatomy.—The mucous membrane of the bronchi in chronic bronchitis is smooth, shining, and of a slatey gray or dark purple colour, a brighter colour being often present in post-mortem examinations, due to an acute exacerbation which so often causes the fatal result. Closer examination will show the open mouths of the dilated mucous glands like pin-point depressions. The mucous membrane is often thrown into folds by the accumulation of cellular structures beneath the basement membrane, and the epithelium consists either of the single layer of flat germinating cells or heaped-up collections of transitional cells, fully formed ciliated epithelium being generally absent. The smaller bronchi are filled with sticky yellow muco-purulent secretion which oozes from the open mouths of the tubes on section of the lung. In uncomplicated chronic bronchitis the muscular coat is hypertrophied, and the tunica adventitia thickened from cellular infiltration, which also extends to the peri-bronchial and peri-vascular fibrous tissue. The cartilages are usually atrophied, or have disappeared entirely. In old patients they may undergo calcareous degeneration. The lungs are usually emphysematous, and in very chronic cases with dilatation of the bronchi the muscular coat may have disappeared. The mucous membrane may be ulcerated in fœtid bronchitis and in bronchiectasis. The lymphatic glands are invariably enlarged and pigmented, and frequently caseating. The heart, and especially the right ventricle, is generally dilated; and the liver, spleen, and kidneys are chronically congested, and the seat of fibroid degeneration.

Symptoms.—The symptoms vary considerably in different cases, but mainly resemble in character those of the acute form, especially modified by coexistent affections. In the milder forms of the common winter cough of old people at first the only complaint is cough with muco-purulent expectoration and slight dyspnœa on exertion, or in the early morning, unless there is marked co-existent emphysema. There is no pain or fever in the earlier stages. With the return of warmer weather the attack passes off completely, only to return with the recurrence of cold and changeable climate. The affection may continue in this manner for several years, but gradually dyspnœa on the slightest exertion is noticed as the attacks increase in severity, and the cough, instead of disappearing during the summer months, tends to persist all the year round, the incessant cough and purulent expectoration disturbing the patient's sleep, the patient's health and strength being gradually undermined.

In the young and in healthy adults the milder cases generally subside gradually with complete convalescence; but when once chronic bronchitis has become pronounced and well established, the pathological structural changes which arise render complete recovery scarcely possible.

Physical Signs.—The chest may be moderately distended without alteration in shape; but patients with old-standing chronic bronchitis are almost invariably emphysematous, in which case the chest is considerably distended and barrel-shaped; the respiratory movements are limited, expiration being prolonged; the percussion-note is hyper-resonant and clear; the breath-sounds are harsh and loud; deep-toned rhonchi, high-pitched squealing, piping sibilus, large and small râles, or bubbling, according to the varying conditions of the tubes, are constantly to be heard, while finer crepitation may often be found at the bases.

The expectoration is very variable in quantity, being muco-purulent, and sometimes streaked with blood from the rupture of small bronchial vessels.

Course and Event.—In the milder cases the general health may not be greatly impaired; but in course of time, especially when the lungs have become emphysematous, the heart becomes dilated, and evidence of cardiac failure is shown by chronic gastric catarrh, enlargement of the liver, and in some cases by albuminous urine. These secondary complications are no doubt largely due to the affections which are the real cause of the bronchitis. Various other pulmonary complications, such as bronchiectasis, chronic interstitial pneumonia, are liable to occur, and the patient sooner or later succumbs to a general failure of health and strength, unless an acute exacerbation, which is very liable to arise, or some intercurrent affection, carries him off.

Diagnosis.—The cough and expectoration without consolidation of the lung usually leaves no doubt as to the diagnosis. From pleurisy it is distinguished by the persistence of vocal fremitus and resonance, and respiratory murmur, and by the absence of bulging, and by the presence of the râles; from pneumonic consolidation, by the absence of complete dulness, tubular breathing, and bronchophony.

The conditions which are most liable to cause difficulty in diagnosis are tuberculous deposits, and bronchitis due to the presence of aneurysm or of new growths pressing on the bronchi.

Prognosis.—The prognosis depends mainly on the age of the patient and the coexistence of complications. Once established, it is only in the comparatively young and robust that complete recovery can be hoped for. In older patients the presence of well-marked emphysema, bronchiectasis, or any valvular heart affection must add to the dangers of intercurrent affections or acute exacerbations.

CLINICAL VARIETIES

The condition of the muscle of the right heart is of even greater import than the integrity of the cardiac valves, for the latter defect is often largely discounted by compensatory hypertrophy, whereas a degenerated and weak cardiac muscle is unable to cope with the demand for increased energy which is made on the right heart in bronchitis, and often fails to respond to any treatment.

The above description applies to the great majority of cases of varying severity. Attention has already been directed to the clinical importance of various diatheses and diseases which predispose to bronchitis. Often enough the bronchitis is only a prominent symptom of some such underlying disease, and some writers distinguish various clinical types, such as renal, gouty, syphilitic bronchitis, and so forth.

But there are certain forms of chronic bronchitis which call for special mention, viz. (*a*) *Bronchorrhœa*, characterised by excessive secretion of glairy, semi-transparent matter, like white of egg mixed with water, containing grayish or yellowish-green masses; or the expectoration may be thin, watery, and clear—*bronchorrhœa serosa.* The cough and dyspnœa are usually paroxysmal, either limited to an hour or two on awakening in the morning, or coming on at intervals of several hours during the day, the amount expectorated being very large, sometimes as much as three or four quarts in the course of the day. During the paroxysms, dyspnœa is urgent; but in the intervals, and in fact throughout the day in some cases where the cough and expectoration only occur on waking in the morning, dyspnœa may be absent or scarcely noticeable. Râles and rhonchi are audible before and during the periods of expectoration; but in the intervals, until the secretion has re-accumulated, the auscultation may reveal comparatively few adventitious sounds. These cases are almost invariably associated with some degree of bronchial dilatation.

Patients may continue to live with little alteration in their condition for many years; but gradually the symptoms become more pronounced, the dyspnœa increases with a tendency to asthmatic symptoms, and generally failure of the circulation, increasing œdema, cyanosis, and impairment of health and strength.

(*b*) *Dry Chronic Bronchitis.*—The *catarrhe sec* of Lænnec is characterised by paroxysms of very troublesome and severe cough, with very scanty expectoration of small masses of tough, viscid, hyaline mucus. The affection is almost always complicated by emphysema, and is usually associated with gout. It is generally regarded as due to congestion of the tubes; but "bronchial spasm is doubtless largely associated with the congestion; indeed, bronchial susceptibility and bronchial irritation are its unmistakable etiological factors" (Ewart).

(*c*) *Fœtid Bronchitis.*—In the course of a long-standing chronic bronchitis, the expectoration occasionally assumes almost suddenly, and without apparent cause, a dirty gray colour with a peculiar putrid odour. Putrid expectoration is met with in bronchiectasis, gangrene, and other destructive lesions of the lung, and in perforating empyema, and is usually due to one or other of these affections; but rarely it supervenes in uncomplicated cases of bronchitis. Its onset is generally attended with rigors, and with all the appearance of the occurrence of acute bronchitis superadded to the chronic affection, with fever of typhoid character, and attended with intense depression. The expectoration consists of a grayish-white alkaline, putrid, muco-purulent fluid, with a peculiar, sickly, characteristic odour which is said to suggest the smell of acacia blossoms. The amount secreted is very considerable; the sputa separate into an upper fluid layer covered with froth, and a lower dirty layer containing yellowish plugs varying in size from a millet-seed to a bean, "Dittrich's plugs." These masses on examination are found to be made up of pus-cells, oil-globules, fatty acids, leucin and tyrosin, and detritus. Various micro-organisms have been isolated from the plugs, leptothrix pulmonalis (Leyden and Jaffé), a short slightly curved bacillus which on culture gives an odour like that of the sputum (Lumniczer), and short thick rods resembling bacillus coli (Hitzig). Virchow and Gamgee observed that these masses stain blue with iodine.

In the milder forms when the patient's strength and general condition is good, the prognosis is fairly favourable, but in severe cases it is liable to lead to various dangerous complications, such as pneumonia, bronchiectasis, gangrene, etc., with usually a fatal result. Death has occurred in some cases from the formation of metastatic brain abscess, but more usually the patients succumb to a general depression and collapse.

(*d*) *Plastic Bronchitis.*—The special feature of this peculiar and rare form of bronchitis is the expectoration of branching casts of the smaller bronchial tubes. Somewhat similar casts or moulds of the bronchi may be found by extension downwards of membranous laryngitis, and are occasionally found in pneumonia, phthisis, erysipelas, and other diseases, or as the result of severe irritation by the inhalation of steam, ammonia, etc.; and fibrinous blood-casts may be expectorated in hæmoptysis. From all these conditions true plastic bronchitis differs in pathology and symptoms.

Etiology.—The cause of the affection is not known, and it is possible that in different cases the etiology is not identical. Its appearance seems to be due to some idiosyncrasy or peculiar features on the part of the sufferer. It has often been associated with a predisposition to tuberculosis, but it may attack those apparently in robust health. The exciting causes of the attacks are much the same as in ordinary bronchitis, generally occurring in the early spring months and after exposure to cold. It is nearly twice as common in males as in females, and, while it may be observed at all ages, is most frequent between twenty and forty.

Symptoms.—The majority of cases are of the nature of a chronic bronchitis, with special features, but, especially in children, acute attacks lasting from one to four weeks occur. Usually the onset resembles simple bronchitis, with a dry cough or with slight mucous expectoration, slight constitutional disturbance; after a variable period, with the formation of the fibrinous casts, severe hacking cough, dyspnœa, rapid breathing, and pyrexia supervenes, followed by the expectoration of the arborescent moulds.

The sense of suffocation and lividity may be pronounced if the bronchi are extensively implicated, and the patient frequently complains of pain in the side, but with the expectoration of the casts there is generally an immediate temporary relief. Expulsion of the casts is followed by hæmorrhage from the bronchi, varying in amount from a few streaks to several ounces. The casts appear in the sputum rolled into a solid mass, mixed with ordinary muco-purulent matter; but when placed in water the casts unroll and display their characteristic form.

In severe cases the interference with respiration is so considerable that total suffocation may ensue before the casts have been expelled. In other cases the course of the affection is less severe, and the attacks may last for weeks or months, or recur at intervals for many years.

The illustration on p. 120 is from a specimen of those finely branched fibrous casts in the Museum of the Royal College of Surgeons, London. They were expectorated by a boy aged eleven. He had always been delicate, and when about six years old had an attack of influenza. From that time he was subject to cough and expectoration, spat up pieces of membrane at intervals. His mother's family was healthy; but on the father's side there was a strong tubercular tendency, and two of the six children had died, one of croup, one of consumption.

The physical signs are those of severe bronchitis, together with those resulting from more or less extensive occlusion of the bronchi. The casts never extend to the trachea, and rarely exceed a goose-quill in size. If only a few smaller bronchi are involved, special physical signs may be imperceptible, but usually there is absence of breath-sounds over the implicated areas, firstly from the blocking of the bronchi, and secondly from collapse of the corresponding air-vesicles. If the area involved is extensive, there may be dulness on percussion here; either hyper-resonance elsewhere, or the respiratory movements may be diminished, and retraction of the lower chest-wall may be present during inspiration. As the casts become loosened, râles or sibilant or whistling bronchi may appear, and a flapping sound has been observed.

The casts vary in length, from being mere fragments, to as much as four or five inches, or even, as in a case of Riegel's, six inches; but generally they are one or two inches long, and when washed free from adherent mucus and suspended in water, form a perfect reproduction cast of the part of the bronchial tree. The larger stem is less in circumference than the tube in which it is formed; it rarely exceeds a goose-quill in size; and following the subdivisions of the bronchi, extends downwards to their finest ramifications, so that, according to Biermer, the minutest terminations may be bulbous from being moulded in the infundibula. Excepting the smaller filaments, the casts are hollow, the lumen being usually filled with mucus and bubbles of air. A transverse section shows that the casts are evidently deposited in successive layers, for they are always found to consist of concentric laminæ of a fibrillated or hyaline basis, with numerous epithelial cells, leucocytes, oil-globules, granular débris, occasional Curschmann's spirals, and Charcot Leyden's crystals in its meshes; blood-cells may be found on the surface. The disease is commonly termed fibrinous bronchitis, under the impression that the casts are composed of fibrin; but it has been shown by Grandy that they are composed of mucus, and not of fibrin, and are analogous to the casts of mucus colitis. Wilson Fox states that they are soluble in alkalies and in lime-water.

Treatment.—Little can be said in favour of any special drug or method of treatment in these cases, but during the attack the patient should be placed under the general conditions and ordinary treatment of acute bronchitis. Inhalations of ammonia, of finely sprayed lime-water, of solutions of an alkaline carbonate, and the internal administration of iodide of potassium, creasote, turpentine, are said to have been employed with success.

Emetics may favour the expulsion of the casts, and Osler suggests pilocarpine might be useful, as it increases the bronchial secretion.

In the intervals between the attacks general hygienic measures and the use of tonics may perhaps prevent the attacks being so trying to the patient; but Walshe believes that no drugs, nor the best of health, nor the most favourable climates have any beneficial influence in modifying or preventing the attacks.

The varying phases of chronic bronchitis render it difficult to lay down any definite course of treatment, and the remedies and method of treatment of chronic bronchitis are so very numerous that it is only possible to mention a few, while endeavouring to suggest the lines of treatment that may be most suitable for the great variety of cases in which chronic bronchitis is the main feature. The most essential point is to discover, and, as far as practicable, remove the cause of the bronchitis in any particular case. Thus if the affection is due to the inhalation of irritating particles of dust in his work, it may be necessary for the patient to change his occupation; if it is due to damp and unhealthy surroundings, he must be placed in a more suitable environment; if to Bright's disease, renal inadequacy, valvular heart-affection, syphilis, or gout, treatment appropriate to these diseases is essential for relief.

In the milder and more chronic cases reliance should be placed mainly on general hygienic treatment, avoiding when practicable the use of drugs, unless specially indicated or required for the purpose of overcoming any complication or constitutional taint. But acute or subacute exacerbations generally call for more active treatment on lines similar to that in acute bronchitis.

We may briefly consider the methods of treatment under four headings: (1) General and climatic; (2) medicinal; (3) local applications; (4) counter-irritation, massage, baths, etc.

(1) *General and Climatic.*—The need for warm clothing, with flannel garments next the skin suited to the season, seemingly so obvious, is not always observed, and may have to be impressed on the patient, while the greatest care should be taken to avoid exposure to cold winds and rapid changes of temperature. Abundance of fresh air, either out of doors, or, if that is impossible, in warmed and well-ventilated rooms, is of the first importance. Too often sufferers from bronchitis increase their susceptibility, and even directly maintain their complaint, from shutting themselves up in close, stuffy rooms. Apart from the various affections which may be the essential cause of the complaint, any impairment of the general health tends to prevent recovery, thus any coexisting affection demands attention; especially does this apply to dyspepsia and constipation.

Nothing more generally proves beneficial than change of climate. Many cases do well by the sea-side. In England the south coast furnishes many suitable places in the winter, such as Torquay, Falmouth, Bournemouth, Hastings, the Scilly Isles, Isle of Wight; while the French Riviera, Biarritz, Madeira, or the northern African shore may prove more suitable to those who are able to go farther afield. But it is well to remember that a very large number of persons are always prone to derangement of the liver and stomach at the sea-side, and that this is one of the conditions which it is very necessary to avoid in bronchitis. For these a warm, equable climate such as that of Malvern, Clifton, or dry hill-air, or the Lake of Geneva, Bordighiera, or Egypt may be better suited. Various Continental spas, such as Ems, Soden, Mont Doré, Carlsbad, Spa, etc., are beneficial, especially in gouty cases.

The diet should be light and nutritious, and the state of the stomach and liver, and any tendency to constipation, should receive careful attention.

(2) *Medicinal.*—In the acute exacerbations and in the very chronic cases the secretions tend to be scanty and the expectoration very tenacious. In these conditions, for increasing the expectoration and making it more fluid, iodide of potassium, carbonate of ammonia, apomorphia, ipecacuanha, cocillana, and citrate of potash are useful, *e.g.* ℞ Ext. cocillanæ fl. ♏xx.; apomorph. hydrochl. gr. $\frac{1}{20}$; syr. prun. virg. ʒj.; aq. dest. ad. ℥ss. Tert. quæ. hor. Or, Ammon. carb. grs. iv.; tinct. scyllæ ʒi.; aq. chlorof. ad. ℥ss. Quatt. quaæ. hor.

If there is a tendency to bronchial spasm, iodide of potash, caffein, lobelia, myrtus chekan, grindelia robusta, or bromide of ammonium may be especially indicated. When the cough is excessive and aimless, and due largely to mere bronchial irritation rather than the amount of secretion to be expectorated, some sedative should be combined with the other remedies, such as morphine, codeine, compound tincture of camphor, belladonna, stramonium, hydrocyanic acid, etc., *e.g.* ℞ Ext. liq. grind. rob.; ext.

liq. myrtus chekan, āā. ♏x.-xx.; tinct. lob. eth. ♏x.; aq. chlorof. ad. ℥j. Quatt. quaæ. hora.

When, on the contrary, the bronchitis is associated with profuse mucopurulent expectoration, the various preparations of opium and other direct sedatives should never be given without the most careful consideration, and then only with great caution, and in combination with some stimulant expectorant, such as the carbonate or chloride of ammonium with squill and senega. In these cases there is often considerable general weakness, for which iron, quinine, arsenic, and dilute mineral acids are desirable, especially in those cases in which the expectoration continues to be excessive despite the exhibition of the foregoing remedies, and in these conditions resort may be had to various gum-resins and other expectorants, examples of which are the balsams of Peru and tolu, Canada balsam, ammoniacum, copaiba, cubebs, creasote, tar or tar-water, terebene, turpentine, terpine hydrate, oil of sandalwood, etc., etc. Wilson Fox has recommended the tincture of larix Europœa as a valuable remedy in this class of cases, and also sulphur given internally as an electuary, combined with bitartrate of potash. For the chronic bronchitis of the aged, Whitla has found ammoniacum a most valuable expectorant; it relieves wheezing and promotes expectoration.

Cod liver oil is often singularly valuable in very chronic cases, especially in those attended with profuse expectoration. Not only does it improve nutrition, but its use is often followed by a remarkable diminution in the secretions of the bronchial mucous membrane when all other remedies have been tried without effect. Nor should we forget that the weakness and dilatation of the right side of the heart, with which chronic bronchitis is so often associated, calls for such remedies as digitalis, strophanthus, and ether.

(3) *Local Applications.*—Local applications include various inhalations and sprays, and intratracheal injections. For steam inhalations, benzoin, terebene, oil of Scotch pine, and creasote are commonly employed: thus a fluid drachm of the compound tincture of benzoin may be mixed with a pint of hot water at 104° F. and the steam inhaled. If there is pain or bronchial spasm, two or three minims of chloroform, conium, or laudanum may be added. With the pine oils and creasote it is well to add carbonate of magnesia, a useful formula being creasote, ♏x., or oil of Scotch or Swiss pine, ♏xl.-lx.; light carbonate of magnesia, 20 to 30 grs.; water to 1 fl. oz., for each inhalation. Atomised aqueous solutions of ipecacuanha wine as recommended by Murrell, chloride of ammonium, very weak tar-water, used with a Richardson's or other form of atomiser, or terebene, eucalyptol, menthol, or thymol, dissolved in vaseline oil, may be mentioned, as well as the wearing of a respirator with a small quantity of cotton-wool or sponge upon which has been dropped some of the volatile expectorant.

The value of intratracheal injections by means of a syringe with a long suitably curved vulcanite nozzle, in certain cases merits careful consideration; the method is little practised, but is highly commended by those who have had recourse to it. It is particularly in patients with tenacious mucopurulent expectoration and in putrid bronchitis and bronchiectasis that the method is called for. Guided by a laryngoscopic mirror, the nozzle is gently but quickly passed below the vocal cords and the syringe emptied. Commencing with a drachm for each injection, the patient soon learns to tolerate two or three drachms being thrown in at a time. An oily menstruum, either olive or almond oil, or liquid vaseline, in which menthol, terebene, eucalyptol, or creasote have been dissolved (about three to five grains or minims to the fluid drachm, separately or in combination) forms a suitable injection. Grainger Stewart's formula for bronchiectasis is—menthol, ten parts; guaiacol, two parts; with olive-oil, eighty-eight parts; a fluid drachm being injected daily. Another suitable formula is—chloroform, ♏xxx.; balsam of Peru, ʒii.; oil of eucalyptus, ʒii.; castor oil to ℥i.; half a drachm being injected once or twice daily.

(4) *External Applications, Massage, and Baths.*—One of the most important means of relieving chronic bronchitis is counter-irritation, long continued rather than severe. Of various stimulating liniments, we may mention the compound camphor liniment, turpentine liniment with acetic acid, or a liniment containing cantharides or capsicum, or the application of iodine in solution, or mustard oil will be suitable for the purpose. The vigorous rubbing of the skin in the application of liniments is no doubt in itself beneficial. Nor should we overlook the undoubted value of systematic massage and Swedish exercises in a large proportion of cases. In young adults, cold baths followed by friction with a coarse towel, needle-baths, in fact a course of "hydropathic treatment," may be very helpful in restoring vascular tone and the patient's general health, while materially assisting in the treatment of the bronchial affection.

III. Bronchitis in Children

Etiology.—Bronchitis occurring in children, while essentially similar to the affection in the adult, is nevertheless distinguished by certain predominating clinical features. The actual cause of acute bronchitis in children is usually exposure to cold, but it is in the poorly clad, under-fed, ill-housed children of the poor who are brought up in dirty, ill-ventilated, overcrowded rooms that the worst forms are observed. Individual predisposition largely influences the nature of a catarrhal attack, and while one child is prone to catarrhal affection of the upper respiratory tract, another tends to suffer from bronchial affections, and, again, others from gastro-intestinal catarrh.

Rickets, dentition, and intestinal catarrh are important etiological factors, and, as Ashby remarks, during the time that a tooth is being cut children seem very apt to suffer from catarrh, which in winter affects the bronchial tubes, and in summer the intestines: pressure of the tooth on the gums seems to act reflexly in producing a catarrh, sometimes with more or less spasm as the child becomes wheezy at night, sibilus being heard all over the chest, while in the morning it will be perfectly well.

A not unimportant factor in the occurrence of bronchitis is buccal respiration from nasal stenosis. The susceptible bronchial mucous membrane of children is unable to withstand the habitual inspiration of air unwarmed and unmoistened by normal nasal respiration, and consequently frequently recurring bronchial attacks are generally observed in children with post-nasal growths.

Whooping-cough and measles are generally accompanied by bronchitis, the bronchitis very often attacking the finer tubes and being mainly responsible for the mortality of these diseases in young children.

Symptoms.—Mild, uncomplicated bronchitis in children is attended by much the same train of symptoms as in the adult, but there is a much greater tendency for the catarrh to involve the finer bronchial tubes and the air-vesicles, a condition which is always attended with graver symptoms and considerable risk to life, owing to the weakness of the muscles of respiration (including the bronchial muscle) and the yielding nature of the chest-walls, features which more especially characterise rickets. In slighter attacks the pulse is moderately quickened, and the temperature raised two or three degrees above normal; but whenever the bronchial tubes are extensively implicated, the pulse is hard, the temperature is four or five degrees above normal, the respirations are quick, sometimes amounting to 70 or 80 a minute, with the alæ nasi distended and working. The chest is expanded in the position of inspiration, the shoulders raised, respiration being chiefly abdominal, and the accessory muscles are brought into play; and the air failing to distend the lungs, the dyspnœa is attended with recession of the chest-walls, especially of the epigastric and lower lateral regions. The skin becomes hot and dry, and the child restless and tossing, first to one side and then to the other, or having to be carried about, and constantly changing its position.

The invasion may be ushered in by rigors or convulsions, and convulsions not infrequently occur in the last stages of fatal cases.

It is only the milder cases which escape complications, especially collapse of the lung, broncho-pneumonia, bronchiectasis, and emphysema; but in young children the onset of the attack may be severe, with great prostration, rigors or convulsions, bronchial spasm, pallor and lividity, and pronounced dyspnœa, which may rapidly asphyxiate the child before there has been time for the development of complications.

The pathological processes which result in *collapse of the lung* have already been described above (see p. 121). The symptoms are not very distinctive, unless the collapsed areas are in the aggregate sufficiently extensive to produce obviously increased respiratory embarrassment, falling in of the chest-wall, and dulness on percussion; and even then it is always difficult, if not impossible, to eliminate the coexistence of broncho-pneumonia, which in such cases is itself the chief cause of the severer symptoms.

The occurrence of broncho-pneumonia in the course of acute bronchitis may be suspected from the exaggeration of all the symptoms which usually result. "The child is restless, the cough shorter and more hacking, the skin hot and dry, the evening temperature usually reaching 103° or 104° F. with morning remissions of several degrees, so that the fever assumes a remittent type; the dyspnœa is usually great, the respirations numbering forty or fifty, but varying with the amount of fever and extent of the lung involved" (Ashby). Examination of the chest unfortunately is often of very little assistance in the diagnosis of this complication, for in place of dulness on percussion there is more usually hyper-resonance owing to the emphysematous lung surrounding the pneumonic patch, and it is rare to find complete dulness; indeed a considerable amount of pneumonia may exist in patches without obvious diminution of the percussion resonance. Ashby draws attention to the fact that while rhonchi are heard all over the chest, over the pneumonic portions râles of a consonant or ringing character may be heard, which are more intense and ringing because they travel to the ear through consolidated lung; and he states that even though no consolidated lung can be detected by percussion, the presence of consonant, intensely ringing râles with a temperature of 103° or 104° points almost certainly to pneumonia. Walshe points to the greater diffusion of the moist rhonchi in bronchitis as compared with pneumonia.

The Diagnosis of acute bronchitis in children is, as a rule, easy if suspected and sought for, although there is seldom any expectoration to be seen, inasmuch as young children almost always swallow what they cough up. There may be considerable laryngitis and glottic as well as bronchial spasm, sometimes causing confusion with spasmodic croup, but in bronchitis the spasm is not so marked, and the cough is less brassy and ringing in character. Laryngismus stridulus is a nervous affection, which, like bronchitis, is especially prone to occur in the subjects of rickets; but it is accompanied by other characteristic signs, and is, moreover, unattended with pyrexia, which is never absent in acute bronchitis in children.

Simple bronchitis in children, as in adults, may also be simulated by the invasion of measles, whooping-cough, typhoid fever, generalised tuberculosis of the lung, and more rarely by scarlatina and small-pox. The chief points of distinction have already been mentioned in the section on acute bronchitis in adults, but it is especially in children that difficulty in the earlier recognition of measles and whooping-cough is likely to arise.

Prognosis.—Acute bronchitis is especially fatal during the first two years of life, and, speaking generally, the younger the patient the more likely is a fatal termination, particularly in rickety children. The chief source of danger is the liability to involvement of the finer bronchial tubes, with asphyxia from extensive pulmonary collapse and broncho-pneumonia. Yet children will sometimes recover from a condition that is apparently hopeless. Hilton Fagge states that we must be cautious in giving an unfavourable prognosis in children; it is surprising how rapid may be both the pulse and the breathing, for two or three days together, in those who ultimately recover completely.

Chronic bronchitis in children is comparatively rare, since the processes of repair are more active in them than in adult life, while the very acute and severe attacks of acute bronchitis, from which only incomplete recovery is generally possible, frequently end fatally before the chronic stage is reached. Nevertheless, susceptible and delicate children are very prone to suffer from frequently recurring attacks of acute or subacute bronchitis, which ultimately pass into a more or less persistent chronic affection. In the milder cases they often lose their tendency to bronchitic affection about the age of puberty, and ultimately become strong and robust.

When children are the subject of persistent chronic bronchitis it is almost invariably accompanied by emphysema and bronchiectasis. The chest becomes barrel-shaped, the shoulders rounded and high, the face pinched, and the body thin. The heart is dilated, the jugular veins large and prominent, and more or less cyanosis is usually observed. They are very liable to be carried off by an intercurrent acute attack, and are apt to become tuberculous.

Treatment.—The treatment of bronchitis in children is the same in principle as in the case of adults. The temperature of the apartment to which they are confined should range between 64° F. to 66° F., and in all but the milder cases the air should be kept moist by means of a steam-kettle. A linseed and mustard poultice, or hot fomentations, should be applied at the outset. It is necessary to be careful not to apply poultices or fomentations too hot for the tender skin of children, or to use much mustard in the poultice; and in infants and young children it is well to confine poulticing to the back if there is great difficulty in breathing, as the weight of a poultice may add a fresh danger instead of producing the relief desired.

The drugs most relied on are ipecacuanha in the form of the powder or wine, syrup of squills, apomorphia, and antimony. Small frequently repeated doses of the tartrate of antimony or of apomorphia will generally relieve congestion and aid secretion in the earlier stages. It is undesirable to push these or any drugs to the point of vomiting or even nausea in the early stages. Small doses of aconite or belladonna, a minim or half-minim every two hours, are also of great service at this early period. Belladonna, recommended by Ringer and Murrell, has the effect of diminishing the secretion in the bronchial tubes, and on this account is often of much value in the bronchitis of children, when from the free secretion both into the bronchi and the pulmonary tissues there is a risk of broncho-pneumonia. It is much praised on this account by Coutts in the treatment of broncho-pneumonia, and I am able to confirm his good opinion of it from my own experience. The drug should be given with no timid hand, the extract in doses of gr. ⅛ to ½ being preferable to the tincture, but its effect must be watched, as it sometimes fails to relieve the respiratory embarrassment. If cough is excessive, small doses of bromide of potassium may be given. If opium or any of its preparations are considered necessary, they must be used with great caution. Non-depressing emetics such as ipecacuanha or sulphate of zinc are sometimes called for in the initial stages before the secretion has occurred, when the breathing is much impeded from the swelling and engorgement of the mucosa; they then may relieve the condition by aiding free secretion and by emptying an overloaded and distended stomach. Again, in later stages, when the tubes are becoming choked by excessive secretion which the little patient, either from weakness or from the tenacious character of the mucopus, is unable to cough up, emetics are of signal service.

The diet should be light and nourishing, such as beef-tea, milk and milk-puddings, Benger's food, and barley-water, and in all cases the condition of the stomach and bowels should receive attention.

The importance of warm clothing, fresh air, and protection from cold and damp as prophylactic measures is obvious, and only requires mention.

Insanity of Cyanosis

A brain supplied with only partially aerated blood, like other organs of the body under similar conditions, is incapable of perfect functional activity, and the greater the coexistent defects in the brain structure the more pronounced is the functional aberration. Thus it is easy to understand that it is especially in persons of advanced years that mental obscurity, confusion and delirium, and various intellectual and emotional phenomena are apt to arise from prolonged or acute cyanosis in bronchitis and other pulmonary affections, and in various forms of cardiac disease. Clouston states that "the insanity of cyanosis from bronchitis, cardiac disease, and asthma is a form of delirium, with confusion, hallucinations of sight, sleeplessness, sometimes suicidal impulses and vague fears. These symptoms are usually worse at night, and often end in mental torpor, passing into coma, and in some degree the mental power is usually affected in most old persons with diseases that prevent the blood being properly oxygenated."

But there are minor degrees of mental disturbances also due to cyanosis which do not amount to actual insanity, evidenced by change of manner, emotivity, suspicion. Maudsley, in discussing mental changes due to impurities in the blood, distinguishes three stages—firstly, a general disturbance of physical tone; secondly, the engendering of chronic delusions; and, thirdly, in the more acute forms, active delirium. In the aged the cyanosis and dyspnœa of capillary bronchitis cause muttering delirium or delusions, but in young, strong adults acute maniacal excitement is sometimes observed.

From the insanity of cyanosis we must distinguish those curious cases in which "alternation" between insanity and, for instance, bronchitis or asthma takes place. "There are certain cases of chronic bronchitis so-called which, by their periodicity and by their chronicity, appear to have a distinct relationship to nervous disorder; and, in confirmation of this, we would say that just as certain cases of asthma alternate with other neuroses, so these cases of chronic bronchitis alternate with the neuroses. Asthma may be distinctly found to alternate with insanity; as a rule the insanity with which it alternates is of the melancholic type" (Hack Tuke). The same alternation is sometimes observed in chronic bronchitis of the type just mentioned, and Tuke refers to one such case in whom there had been two attacks of melancholia, and each of these was associated with freedom from the recurrent bronchitis.

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Bronchiectasis

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DEFINITION. — Bronchiectasis or dilatation of the bronchial tubes is secondary to, or occurs as an incident in a variety of pulmonary diseases. In some cases the condition is masked by the primary disease and unrecognised during life. This is especially the case in the acute forms when the smaller bronchioles are dilated (bronchiolectasis), and in the chronic form of the disease where there is general and fusiform dilatation of many tubes.

In the more typical cases the disease is easily recognised by certain prominent and characteristic features which entirely obscure the primary disease.

ETIOLOGY AND GENERAL CAUSATION.—In the histories of patients who are the subjects of bronchiectasis we find the following diseases:—

1. Acute or chronic inflammatory diseases of bronchi, lungs, or pleura.
    (*a*) Acute and chronic bronchitis with or without emphysema.
    (*b*) Broncho-pneumonia, acute and chronic pleuro-pneumonia.
    (*c*) Chronic pleurisy and empyema.
2. Obstruction, compression or stenosis of bronchial tubes.
    (*a*) Obstruction by foreign bodies in the bronchi.
    (*b*) Compression by aneurysm, hydatid, malignant, glandular, or congenital tumours (*e.g.* dermoid cysts).
    (*c*) Stenosis as a result of syphilitic or other ulceration, with consequent stricture.

3. Chronic lung-diseases (tubercle, cancer, etc.) producing secondary bronchiectases, partly by inflammatory process, partly by compression.

In some cases, however, no previous history of disease can be traced, and it has been suggested that some congenital weakness of the bronchial wall or deficient elasticity of lung has existed.

Much discussion has taken place upon the mechanical cause of dilatation of bronchi, and without entering fully into the many theories propounded, it may be taken for certain that two general conditions are usually present:—

1. Some weakening of the bronchial wall, either congenital or more commonly of inflammatory origin.

2. Some alteration in the normal conditions of pressure either within or without the tube.

Inducing Cause.—There is no doubt the forced *expiratory efforts* associated with coughing are an important factor in bringing about the yielding of an already weakened wall, though it is impossible to eliminate the effects of dyspnœa with increased *inspiratory effort.* The accumulation of secretion combined with the effects of gravitation doubtless have their due share in some cases.

Morbid Anatomy and Pathology.—The leading features will be readily appreciated by reference to the points indicated under Etiology. Thus the appearances vary with the cause, the duration, and the presence of complications (pyæmia, hæmorrhage, etc.) Two general forms are recognised: (1) *the cylindrical or fusiform;* (2) *the globular or sacculated form.* The bronchial walls may be attenuated and atrophied, and the surrounding lung tissue rarified or emphysematous. More commonly the walls are markedly thickened, the peri-bronchial tissue indurated, the surrounding connective tissue showing a true interstitial and reticular cirrhosis, while the intervening alveoli contain catarrhal products, which later become organised. The chronic pneumonia thus induced is secondary to the affection of the bronchus. In other cases the lung consolidation is primary, and the bronchiectasis secondary. Thus a twofold induration of lung tissue occurs, sometimes confined to the neighbourhood of the dilated bronchus and presenting irregular patches of consolidation, at others spreading to the entire lobe. In acute bronchiectasis the whole lung may be riddled with cavities. This is well seen in children when the lung is often sponge-like and honeycombed. In the fusiform variety long cavities are found, either radiating from the root of the lung to the periphery, or confined to one or both bases. In the saccular form the cavity is usually basal, and it may be deep-seated or superficial, containing very offensive, purulent secretion. In rare cases the dilatation is apical, and is thus usually associated with tuberculous disease. Occasionally a bronchiectatic cavity may closely simulate a phthisical one, but the two can be differentiated by the presence of the basement membrane in the former. The other viscera—heart, liver, brain, kidneys, etc.—may show secondary changes which are, however, not characteristic of this condition. Thus we may find any or all of the pathological appearances of pyæmia, acute or chronic. Hæmorrhages of serous membranes, acute pleurisy or pericarditis, empyema, and metastatic abscesses of brain or other distant parts are not uncommon.

Character of the Secretion and Bronchial Contents.—On standing the secretion forms two layers, an upper frothy, yellowish or brown in colour, and a lower consisting of purulent and solid matter. The odour is extremely offensive and must be smelt to be appreciated. Fœcal is the term that most nearly describes it, but a smell is difficult to define. The solid matter consists of pus-cells, granular matter, and micro-organisms. Crystals are sometimes found of leucin, tyrosin, cholesterin, and fatty acids with many oil-globules. Post-mortem, the dilated bronchi are often found to be filled with pultaceous, caseous-looking material (see "Expectoration").

Clinical Phenomena.—Acute Bronchiectasis or bronchiolectasis is usually a disease of children. Its symptoms cannot be distinguished during life from those of the capillary bronchitis with which it is associated.

Chronic Bronchiectasis is a disease of children or of early or middle life, occurring more frequently in men than in women. When secondary to phthisis, cancer, or obstruction of bronchi from any cause, it may occur in either sex or at any age.

Cylindrical bronchiectasis is only to be differentiated from the chronic bronchitis or other disease of which it forms a part by the excessive bronchial secretion, occasionally by fœtor of the cough or expectoration (though this is by no means constant), and by the enlargement or clubbing of the terminal phalanges of fingers and toes, which is frequently but not invariably seen.

The characteristic symptoms of the disease are more commonly observed in the *sacculated bronchiectases,* less often in the cylindrical. Cough is a prominent symptom, and expectoration with excessive secretion of an extremely offensive and fœtid odour.

The cough is frequently intermittent in character and occurs in violent paroxysms, somewhat resembling whooping-cough, and terminating in the expulsion of a large quantity of mucus or muco-pus presenting the characteristic features described. Sometimes one fit of coughing, or perhaps two, occur during the twenty-four hours, often induced by change of posture (such as rising from bed, stooping, lying down). It seems probable that the reason for the paroxysmal nature of the cough and large expulsion of bronchial secretion is due, in the few cases in which it is present, to the mechanical position of the cavity or cavities, which, when filled beyond a certain point, overflow on the least change of position into the neighbouring undilated tube, and set up an irritable spasm which leads to violent expiratory efforts and subsequent expulsion of the contents.

The sacculated tubes being thus emptied entirely or partially, the patient may be free from cough for the rest of the day and comparatively comfortable. In other cases the cough does not exhibit this paroxysmal nature, but is frequent, though still accompanied by excessive and often offensive expectoration. In children the attack is often mistaken for vomiting. In reality a slight cough is followed by retching and the sudden expulsion, not of vomit, but of fœtid mucus, sometimes exceeding half a pint in amount. *Hæmoptysis* is not at all uncommon, but except in late stages of the disease is rarely excessive. The *temperature* of the patient is not raised unless the disease is associated with bronchitis or catarrh. A frequent though not invariable feature is the *clubbing* of the distal phalanges of fingers and toes. In rare cases other joints share in the enlargement (see "Osteo-Arthropathy, Hypertrophic Pulmonary.")

Nutrition is often well maintained, and the strength and appetite good. In later stages there may be wasting, excessive dyspnœa, anorexia, or alimentary troubles. None of the above symptoms are constant. There may be little or no cough or expectoration. Fœtor of expectoration, cough, or breath may be present or absent. Clubbing cannot be regarded as pathognomonic of the affection, though it is commonly present. In the later stages of the disease grave symptoms are often associated, due to catarrh or broncho-pneumonia, or to ulceration of bronchial tubes. In the latter case acute septicæmia or one of the many manifestations of pyæmia may ensue. Death may occur from—

(1) Acute bronchitis or broncho-pneumonia.

(2) Cardiac failure.

(3) Exhaustion often induced by vomiting or other alimentary affections.

(4) Amyloid disease and its accompaniments.

(5) Hæmorrhage from ulceration into or aneurysm of pulmonary vessels.

(6) Acute or chronic pyæmia with inflammatory or suppurative affections of serous membranes and metastatic abscesses, of which the most frequently recorded are abscesses of brain.

Physical Signs.—There are no pathognomonic signs by which we can distinguish dilated bronchi.

It will be remembered that in the acute cases in children and in some chronic cases of cylindrical bronchiectases, the signs are those of the associated bronchitis only. For the sake of simplicity the physical signs may be described under the following heads:—

1. *Where the disease is masked by the deep position of the Dilated Bronchi or by Emphysema, and unaccompanied by surrounding Pneumonia.*—The chest may be resonant or hyper-resonant (in the case of emphysema). If the cavity approaches the pleural surface there is Skodaic resonance or absolute dulness according as the bronchial dilatation is filled with fluid secretion or not. In the former case breath-sounds at the base or bases (which are usually affected) are absent or feeble, and the voice-resonance diminished. When the dilatation is empty, the respiratory murmur becomes broncho-cavernous, or cavernous with marked pectoriloquy. This variability in the physical signs is important and will be referred to later.

In the cases of deeply seated dilatations it is not uncommon to find that resonance, vocal fremitus, voice sounds, and respiratory murmur do not depart from the normal, or perhaps the breath-sounds are somewhat harsher than usual. Adventitious sounds may or may not be present. When present they vary from the small bronchitic crepitation to the large bubbling râle. In this and the next group of cases, however, a marked feature may be the extraordinary variety of size and sound of râle. When a cavity is superficial, bubbles and râles of every size, squeaking and "croaking" sounds, or merely sibilant or sonorous dry sounds may be present. The term "croak" applied by Ewart well defines the characteristic quality of the sound.

2. *Cases where the Dilatation of Bronchi is associated with a secondary Consolidation of Lung.*—Here the signs of consolidation, either patchy or confined to one or both bases, are superadded. Dulness to percussion, diminished or increased vocal vibration and resonance are present, varying with the condition of the pleura and with the depth of the dilated bronchus. When the case is well marked a feature is the *variation* in the degrees of increased voice-resonance as the stethoscope is passed from one spot to another at the base. In a tuberculous cavity near the surface there is frequently found a pectoriloquy of fairly uniform character and intensity over the whole surface of the cavity. It is not so where a series of bronchiectatic cavities occur at various distances from the pleural surface. Pectoriloquy of varying degree of pitch and loudness is heard at different spots on the chest-wall.

Further, as in the first class of cases, the physical signs vary from time to time with the full or empty state of the bronchial cavity, provided it is near the surface. Again, râles may be absent or numerous, and those that are considered by some authorities to be peculiarly characteristic of the disease are sometimes present, especially the "croak" previously referred to (described under different names by various authors), and a sound known as the "veiled puff" of Skoda, heard in rare cases at the end of inspiration. It cannot be positively affirmed that these two sounds are never heard in other pulmonary diseases, but it is certain that they are more often associated with bronchiectasis.

3. *Cases in which the Consolidation or other Disease of Lung is primary, and the Dilatation of Bronchi a secondary result.*—Space will not allow a detailed description, which would indeed be an account of the physical signs of condensation or fibrosis of lung due to an old pleurisy or empyema, a chronic pneumonia, tuberculous fibrosis, or any indurative pulmonary disease; or, again, of the signs of obstruction of bronchus or of some tumours, malignant or otherwise, producing compression.

Retraction of the side and displacement of the heart are usually the result of a contracting fibrosis or of an old pleurisy, and are not found so frequently in the second group, where the organising pneumonia is secondary to the dilatation of bronchi. When the disease is confined to one or both apices of the lungs the case is usually a tuberculous one, though this is not an invariable rule.

Diagnosis.—(*a*) *From Tuberculous Disease of Lungs.*—In the early stages the comparative well-being of the patient, the absence of wasting and of pyrexia, and the common association of clubbing of fingers (more rare in tuberculosis) serve to distinguish the case from one of tuberculous disease. In phthisis a basic disease, moreover, is of extreme rarity, unless the apex has been primarily affected. Tuberculous cases, however, do occur with fibroid consolidation of one base. These are usually of pleuritic origin, or are found in rare cases of senile tuberculosis.

The absence of tubercle bacilli is to be assumed. It must not be forgotten, however, that bronchial dilatation with its characteristic symptoms is occasionally an accompaniment of tuberculous disease, especially in those forms where there is much induration. The diagnosis is more difficult when there is bronchiectasis with chronic pneumonia of the upper lobe, especially if hæmoptysis be also present. In such cases the physical sign (upon which stress has been laid) of the variability of voice-resonance, and even of respiratory murmur in different situations at the apex, becomes of importance. The changes due to the emptying and refilling of a cavity are sometimes found in a basic tuberculous excavation, but far more often in bronchiectasis. These signs are less likely to occur at the apex of the lung.

(*b*) *The discharge of a localised or small basic empyema* into a bronchus with the formation of a permanent sinus is to be distinguished from bronchiectasis by the history of the disease, and usually by the absence of fœtor of the muco-purulent or purulent discharge. The diagnosis is not always easy, because in an old discharging empyema the fingers and toes are often extremely clubbed, the discharge is intermittent and occasionally blood-stained and fœtid. The fœtor of expectoration is usually a sign that more than a mere sinus is present, and that probably as a result of the pleurisy and consequent cirrhosis of lung some of the neighbouring bronchi have become dilated.

(*c*) *Chronic Bronchitis.*—As we have previously stated, it is often impossible to diagnose dilated bronchi in a case of chronic bronchitis, though it may be conjectured that the condition is present if the sputum becomes fœtid and the fingers clubbed. Most authors describe a form of bronchitis with offensive sputum under the name of fœtid bronchitis or bronchorrhœa. The writer is of opinion that this disease is a myth, and that fœtor of the bronchitic sputum in cases of chronic bronchitis means nothing more than that some of the bronchi are the subjects of fusiform dilatation, and that owing to the accumulation of secretion and consequent decomposition the sputum becomes offensive. When the fingers are not clubbed, care must be taken not to confound fœtor of breath, cough, or sputum with that due to other causes. Pharyngeal mucus is sometimes excessive and of extremely offensive odour, but the discharge is usually in the early morning. Ozæna and the fœtor from carious teeth and the offensive breath from constipation with disturbed liver must all be eliminated.

(*d*) *Gangrene and Abscess of Lung.*—*Gangrene* is an acute and grave condition, and could only be confounded with the later and ulcerative stages of bronchial dilatation. As a rule, the history aids the diagnosis.

The gangrenous odour is usually distinct and characteristic, but may be confused with that of bronchiectasis. Some aid is also to be obtained from an examination of the sputum. In gangrene there is more destruction of tissue, and elastic fibres are frequently found, whereas the expectoration from a bronchiectatic cavity rarely contains them. If gangrene is associated with tuberculosis, tubercle bacilli will also be present. As in the case of gangrene, the formation of a *pulmonary abscess* is sudden in its onset. Whether a sequela of pneumonia or a result of pulmonary embolus, the disease is acute, and usually an evacuation of pus is followed by a quiescence of symptoms.

Cases, however, occur of a basic pulmonary abscess in which fœtor of expectoration, intermittent cough, and discharge of pus and clubbing of fingers are present. They are extremely difficult to diagnose from a sacculated bronchiectasis. The fœtor is not so pronounced as in gangrene. Several cases have been recorded in which operative procedure has resulted in complete cure. A small localised empyema discharging through the lung, or a sub-pleural chronic abscess, may easily be confounded with a single sacculated dilatation of a bronchial tube.

PROGNOSIS.—Bronchiectasis is usually an extremely chronic disease. In children the acute cases associated with capillary bronchitis rarely recover. The condition of the heart and circulation (especially right heart) is an important element in prognosis. The onset of pyæmia, acute or chronic, profuse hæmoptysis, or cerebral symptoms are of grave significance, and usually precede a fatal termination. In addition to the many complications of the disease already referred to, death may result from exhaustion.

TREATMENT.—General Considerations.—Attention must be paid to the nutrition and strength of the patient. A dry, warm, or equable climate, and remedies that improve the appetite and nutrition are advisable. Amongst such remedies may be included cod liver oil and malt extracts, the hypophosphites and glycerophosphates, with or without one of the following tonics: quinine, iron, or strychnine. Two drugs appear to act in a twofold manner. Benzol in five to ten minim doses in a flavoured emulsion, and creosote in capsules of one, two, or more minims, not only increase appetite and improve the digestion, but probably act as antiseptics. They are both easily tolerated after the first few doses. Any or all of these, especially if combined with the influence of a dry, pure air, increase the vigour and recuperative power of the patient, and probably lessen the tendency to catarrh.

Complications.—Bronchitis and catarrhal affections of the lungs must be treated on general principles. *Cardiac failure* will need the administration of digitalis or cardiac tonics. Care should be taken in the case of systemic engorgement to encourage the action of the liver secretion by administration of small doses of mercury or cholagogue purgatives, and to promote diuresis. *Alimentary troubles* are to be dealt with by sedative treatment or digestive tonics, with bland, unirritating nourishment. If *hæmoptysis* occurs, absolute rest in the recumbent position should be prescribed, unless the symptom is very slight. In case of profuse hæmorrhage, morphia may be given with caution, combined with remedies that diminish arterial tension, while the amount of fluid in the diet must be diminished.

Special Treatment.—The object is to diminish bronchial irritation and inflammation and to promote antisepsis.

Guaiacol is of special value when given in doses of 3 to 5 grains several times a day in capsules or cachets. Terebene (5 grains in capsules), oil of turpentine (10 to 20 minims with mucilage in an emulsion), benzoin (10 to 20 minims of the compound tincture), or benzoic acid (5 grains), ammoniacum or guaiacum (half to one ounce of the mistura ammoniaci or mistura guaici)—all these are found to be of service in diminishing chronic catarrh and in lessening the bronchorrhœa or excessive secretion.

Many of these remedies act directly through the blood, and some, like guaiacol, are probably eliminated through the respiratory mucous membrane. If large cavities are present in which secretion accumulates and decomposes, it is of the utmost importance that all the means at our disposal should be adopted to empty these cavities as frequently as possible. Without this the value of astringent treatment is diminished, and all the subsequent measures that are now to be described will be of little value.

Methods of emptying Bronchiectatic Cavities.—Change of posture will often produce violent cough with the expulsion of a large amount of expectoration. In some cases the act of lying down, stooping, or even leaning out of bed is sufficient. The occasional paroxysmal attacks are rather to be encouraged than not.

The Continuous Inhalation of Creosote.—The continuous treatment by the inhalation of creosote has found great favour, and is probably more successful than any other method in bad cases. The patient should be placed in a special room or chamber, in which an ounce or two of ordinary commercial creosote is vaporised on a small metal bath heated by a spirit lamp. The patient's eyes are covered, and the nostrils plugged with cotton-wool. He inhales the dense fumes of creosote vapour for a quarter of an hour to twenty minutes, at first every other day. Ultimately he should be subjected to it for one hour to one hour and a half daily by slow increments in the length of treatment. Tolerance is soon established, and in many cases the relief is considerable. Violent cough and more or less complete emptying of the cavities is the result. Probably some absorption of the drug and possibly a certain amount of local effect are produced, though this is doubtful.

Occasionally the amelioration is rapid and decided, but in many cases the result of months of treatment is only a diminution of expectoration, and especially of the fœtor. Occasionally brilliant results have been produced. It may be asserted that better results have been obtained at the Brompton Hospital from the prolonged use of creosote inhalation than from any other form of treatment.

Provided the dilated bronchi can be partially or wholly emptied, other methods of treatment which are believed to have a local effect may be valuable. These are (1) *Moist Inhalations.* Creosote, turpentine, carbolic acid, or compound tincture of benzoin are mixed with hot water at a temperature of 140° in an inhaler. It is well known that though these remedies are deeply inhaled they do not reach far down the trachea. The plan of treatment is well recognised, but is not of permanent value.

(2) *Intra-laryngeal Injections.*—A 2 per cent solution of guaiacol or of creosote in menthol and olive oil injected daily or every second day is beneficial in some cases.

(3) *Treatment by Drugs which promote Antisepsis and diminish Fœtor.*—Guaiacol and creosote have already been mentioned. Garlic, 1 to 3 grains in cachet, or 1 to 4 drms. of the syrup, either alone with equal quantity of syrup of tolu, or in combination with creosote and syrupus picis liquidæ, has also been recommended. The disagreeable odour following its use is a disadvantage.

Operative procedures have not been attended by good results on the whole. The opening and drainage of cavities at the base of the lung have been frequently tried, usually when it was thought that there was a single cavity. The results are not often encouraging, because frequently multiple cavities have been found where one has been diagnosed. There is some danger of septic absorption into the surrounding tissues and the edges of the wound and sinus formed, owing to the putrid character of the evacuated secretion.

Indications for operation are found in the following signs:—(1) There must be evidence of a *single* cavity. (2) Given a single cavity, high fever and signs of ulceration of bronchi or of putrid absorption completely justify an operation, consisting of resection of portion of one or two ribs and the drainage of the cavity. Complete recovery has sometimes resulted.

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Buhl's Disease. *See* NEW-BORN CHILD.

Bulbar Paralysis. *See* PARALYSIS.

Bunion. *See* DEFORMITIES.

Burns and Scalds.

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THE term *Burn* is applied to the injury resulting from the application of dry heat to the tissues of the body; while the damage done by moist heat is usually spoken of as a *Scald*. The distinction is unimportant, as the essential characters of the lesions produced by the two forms of heat are for all practical purposes the same; and conditions almost identical frequently result from the application of such strong caustic substances as fuming nitric acid, caustic potash, nitrate of silver, or arsenical paste.

CAUSES.—Fire and steam play such an important part in everyday life and work that the accidents producing burns and scalds are infinite in their variety. Among the commonest are the ignition of clothes, especially those of women and children, the bursting of paraffin or other lamps, the spilling of hot fluids, molten metal, or boiling oils, explosions of gas or gunpowder, and the escape of steam in boiler explosions. Severe burns sometimes follow the application of very hot poultices to young and debilitated children.

Such substances as hot solids, molten metal, and strong caustics are local in their action, and produce burns which, although deep, are limited in extent. Flames, exploding gases, and vapours, on the other hand, occasion more widespread injury to tissues, but the damage is comparatively superficial. Their risk, however, is accentuated by the inhalation of poisonous fumes or solid particles of carbon and dust into the air-passages and lungs.

Scalds resulting from boiling water or steam are less severe locally than those caused by boiling oils or saline fluids; but the inhalation of steam into the air-passages introduces additional risks in the form of œdema glottidis, bronchitis or broncho-pneumonia, complications which may also follow burns of the mouth or pharynx resulting from drinking very hot fluids or caustic substances.

Since *electricity* has come to assume such a prominent place in industrial undertakings, a distinct form of burning has been recognised. Mery and Doubousquet-Laborderie have studied this variety of burns, and have formulated the following propositions regarding them: (1) They may be superficial or deep; (2) they are not painful; (3) they are not accompanied by fever, or followed by any constitutional disturbance; (4) being aseptic they do not tend to suppurate; and (5) they cicatrise quickly and well.

Risks.—The main factors upon which the risk of a burn depends are—(1) Its *extent.* It has been shown that burns implicating from one-half to two-thirds of the entire surface of the body are almost invariably fatal. (2) Its *situation.* Burns over the serous cavities of the body—abdomen, thorax, or skull—are, *ceteris paribus,* much more dangerous than those on the limbs. (3) *The age of the patient.* Although young children succumb to the shock of severe burns more readily than adults, they appear to withstand prolonged suppuration better. (4) The presence or absence of *sepsis* is a most important factor in regard to prognosis.

Classification of Burns.—The classification most generally accepted is that of Dupuytren, which has for its basis the depth to which the injury has penetrated. He distinguishes six degrees—I. Hyperæmia or Erythema; II. Vesication; III. Partial destruction of the thickness of the true skin; IV. Destruction of whole thickness of the true skin; V. Charring of muscles; and VI. Charring of bones.

Clinical Features of Burns

General Phenomena.—The severity of a burn depends chiefly on the extent of surface implicated, and to a less degree on the depth of the lesion. The intensity of the heat and the duration of its application are also important factors.

Almost all burns which are of sufficient severity to be brought under the notice of the surgeon present in point of time three distinct clinical stages—I. The stage of *congestion and pain;* II. the stage of *fever and inflammation;* and III. the stage of *suppuration.*

I. *Stage of Congestion and Pain.*—This stage lasts about forty-eight hours, during which the patient is in extreme agony, and the whole area involved in the injury is intensely congested and somewhat swollen. The patient suffers from a form of traumatic shock or collapse, attributable to reflex stimulation of the sympathetic nervous system. As a rule he lies moaning with pain, the face pale, the features drawn or shrivelled, the skin livid and moist with a cold clammy sweat. The temperature is below the normal; the pulse small, weak, and almost imperceptible; and the respirations shallow and irregular. The urine is scanty and high-coloured, and the vital reactions gradually diminish. In cases which prove rapidly fatal the mind is clear at first, and may remain so to the end, or coma may supervene before death ensues. In some cases, especially in children, great mental excitement followed by delirium, cramps, and convulsions are marked clinical features before the fatal issue.

In many cases even of great severity the patient shows a wonderful degree of apathy, lying still, and complaining of nothing except great thirst.

II. *Stage of Fever and Inflammation.*—This stage—the stage of reaction—begins on the second or third day. The pain subsides, the appetite is lost, and constipation, or it may be dysenteric diarrhœa, is present. There is a general congestion of the internal viscera, which may give rise to various clinical complications. Thus *albuminuria* is very constantly present in cases of extensive burning, especially when the temperature is raised to 101° F. or higher. *Respiratory complications* in the form of bronchitis, broncho-pneumonia, or pleurisy are common sequelæ of burns, especially of those occurring over the thorax. Laryngitis and œdema glottidis not infrequently follow scalds produced by the inhalation of steam, or from drinking very hot fluids or caustics.

The *intestinal complications* vary from a slight catarrh with diarrhœa to severe degrees of inflammation and ulceration of the intestinal mucous membrane. Ulceration of the duodenum, leading to perforation, has been observed in cases of severe burning, but its frequency and clinical importance appear to have been exaggerated. It has been ascribed to the digestive action of the gastric juice on the devitalised mucous lining of the gut.

Cerebral complications in the form of meningitis or cerebritis are not infrequent after burns implicating the scalp and neck, and are evidenced by cerebral irritation or delirium followed by convulsions and coma.

III. *Stage of Suppuration.*—This stage begins when the sloughs separate, and its duration and severity largely depend upon the success which attends the efforts made to secure asepsis in the local condition. In severe cases with marked septic infection it may be very prolonged. The patient gradually loses strength, and evidences of septic absorption in the form of septicæmia or pyæmia manifest themselves. At one time tetanus was a not infrequent sequel. Should the suppuration be profuse and long continued, waxy degeneration of the liver, kidneys, or intestines may supervene, and death result from hectic. In this stage death has been attributable in some cases to perforation of a duodenal ulcer.

Local Phenomena.—The description of the different degrees of burns may be prefaced with the remark, that clinical cases almost invariably illustrate more than one degree of burning. The deeper varieties are always accompanied by those of less severity, and the clinical picture is made up of the combined characteristics of all. Further, it may be said that it is often extremely difficult to determine the exact depth of a burn immediately after it occurs; sometimes it turns out less severe than it appeared at first sight, but oftener, perhaps, more so.

Burns of the First Degree.—*Hyperœmia or Erythema.*—These are usually produced by flame in contact with the skin for a very short time; by solids or liquids below 100° C.; or they may result from prolonged exposure to the summer sun's rays.

The pain is acute while it lasts, but it usually passes off in a few hours. The part is of a bright red or purple hue, which disappears temporarily under the pressure of the finger, and which gradually blends with the normal colour of the skin around. There is a transitory swelling of the burned area, with perhaps slight oozing of serum from the surface, and the superficial layers of the cuticle usually peel off later.

Burns of the Second Degree.—*Vesication.*—More prolonged exposure to flames, solids, or fluids at or above 100° C., or direct application of steam, are the common causes of burns of the second degree.

Here and there over an area which presents all the features of a burn of the first degree to an aggravated extent, there appear, either immediately after the accident, or, more frequently, not till some hours later, a number of vesicles or blisters. Each vesicle consists of a raised portion of epidermis, under which may be seen serum of a yellowish or brownish colour. Sometimes the vesicles burst, the serous or sanious fluid escapes, and the surface underneath is seen to be of a bright scarlet colour, the papillæ of the skin standing out as fine velvety projections, excessively sensitive to pressure or friction, and from which serous fluid freely oozes. No permanent scar remains after the healing of such burns, but the part may for some time show a slight depression or dark-coloured pigmentation. Infection by septic bacteria may induce superficial suppuration, and so delay repair.

Burns of the Third Degree.—Partial Destruction of the True Skin.—This usually results from contact with hot metals, the prolonged application of flames, or from fluids at a temperature above 100° C.

The epidermis, Malphigian layers of the skin, and the papillæ are more or less disorganised, and patches may be completely destroyed, leaving hard, dry, and shrunken sloughs of a yellow or black colour, and quite insensitive. The surrounding areas show damage to the first and second degrees. The pain in these burns is intense, but passes off during the first day, to return again, however, when, about the sixth or seventh day, the sloughs separate and expose the nerve filaments of the underlying skin. Granulation tissue fills up the gap, and by its contraction may lead to a certain amount of depression in the pale scar which results.

Burns of the Fourth Degree.—Total Destruction of the True Skin.—Any form of intense heat if sufficiently long applied may produce burning to the fourth degree. The local destruction of tissue extends right through the true skin and usually involves the underlying connective tissue and fat. Large black, dry eschars are formed, around which a ring of white tissue is seen, and outside this a zone of intense congestion which gradually merges into the normal skin. As the cutaneous nerves are completely destroyed in burns of this degree, pain is not severe. Infection is very prone to occur by organisms passing from the surrounding skin into the sloughs, and profuse suppuration often ensues. Cicatrisation is always slow, and leaves deep, irregular, and often puckered cicatrices, which by their contraction in course of time tend to produce varying degrees of deformity according to their situation. In the region of the neck, the face, or the flexures of large joints the distortion is often extreme, and may call for active surgical interference to remedy it.

Burns of the Fifth Degree.—Charring of Muscles.—This degree of burning is frequently met with in epileptic, apoplectic, or alcoholic persons who, while unconscious, have remained for a prolonged period in contact with hot metal, flame, or chemicals. All the tissues of the part down to the muscles are destroyed, and joints or serous cavities may be opened into.

Large nerves or vascular trunks may be implicated, and profuse hæmorrhage may occur, either at the time of the accident, or later when the sloughs separate by suppuration. Septic infection of any synovial or serous cavity which happens to be implicated is more than probable.

In many cases the only hope for the patient lies in amputation.

Burns of the Sixth Degree.—Charring of Bones.—When burning goes to this extent the result is usually fatal, and when a limb is implicated amputation is imperative.

Pathology of Burns.—The cause of death in rapidly fatal, extensive, superficial burns is the chief point of pathological interest. It has long been recognised that the general constitutional disturbance following extensive superficial burns is proportionately much greater than is observed in severe, deep burns. As Bardeen puts it, "There seems to be something especially noxious in the merely superficial burn." Such injuries are almost universally fatal, especially in children, when more than one-half of the surface of the body is involved, even should the burns be of the most superficial kind. Many theories have from time to time been put forward to explain this fact. Thus the views that the grave symptoms resulting so often in death are due to interference with the respiratory, the excretory, the heat-regulating, or even the protective functions of the skin, have each had their advocates. More extended observation by modern methods, however, has thrown doubt upon most of these theories.

The chief naked-eye lesions found after death have been a general hyperæmia of the organs of the abdominal, thoracic, and cerebro-spinal cavities, sometimes accompanied by serous or sanious exudation—conditions which have been attributed to a reflex vasomotor disturbance.

These changes appear to take some time to be produced, because if death be very rapid, they are not observed. If the patient survive a few days, active inflammatory signs may supervene in the intestinal canal, lungs, pleura, kidneys, or meninges, and ulceration may follow in the stomach or duodenum. The relative frequency of these lesions may be gathered from the following statistics of 200 cases by Schjerning:—

Hyperæmia of brain . .	occurred	in	49	per cent.
Hyperæmia of lungs . .	„	„	36·5	„
Nephritis	„	„	28·2	„
Pneumonia	„	„	27	„
Hyperæmia of intestines .	„	„	22·2	„
Pleurisy	„	„	18·8	„
Ulcer of duodenum . .	„	„	12·4	„
Meningitis	„	„	10·5	„

There is also a profound alteration of the blood in these cases. Its specific gravity is increased, the polymorpho-nuclear leucocytes are increased in number, the red cells run together and ultimately break down, and the hæmoglobin set free is deposited in the epithelial cells of the tubules of the kidney, where it produces irritation followed by hæmoglobinuria.

Thrombi and small extravasations are found in many situations throughout the body, probably due to the action of a fibrin-ferment, of which two have been isolated, one a body like muscarin, the other belonging to the pyridine-chinoline group.

Some interesting clinical and experimental observations by Korolenko throw light on these morbid appearances. He found both in patients and animals dying after severe superficial burns, that the ganglion cells of the solar plexus had undergone degenerative changes, varying, according to the severity of the injury, from slight œdema to complete destruction of their protoplasm with disappearance of their nuclei. From his experiments he concludes that the solar plexus is subjected to a reflex excitation, which passes out to the abdominal circulatory system, producing spasm of the vessels, including those supplying the ganglia themselves, whereby nutrition suffers. In the rapidly fatal cases the vessels remain spasmodically contracted, and hyperæmia of the abdominal organs is not observed. In the less severe cases the spasm of the vessels is followed by a passive dilatation which explains the hyperæmia of the viscera. The influence of the solar plexus upon the heart may also be a factor in producing death.

Bardeen has recently observed marked changes in all the lymphatic tissues of the body, from the small nodules of lymphocytes in Glisson's capsule to the largest lymphatic glands—consisting in general œdema of the lymph tissue, especially at the germinal centres, with swelling of the individual cells and breaking up of their nuclei; and the appearance of certain large flattened endothelial cells having a phagocytic action. He also found in his cases marked cloudy swelling of the liver and kidney, and softening and enlargement of the spleen.

The morbid findings throughout, therefore, are so like those met with in diseases where the presence of a *toxine* is known to produce them, that it seems highly probable that death in rapidly fatal cases of burning admits of a similar explanation, although the source and nature of the toxine is as yet undetermined.

Local Histological Changes.—The most noteworthy changes following the application of heat to the surface of the body, are the coagulation of the protoplasm of the cells, and its subsequent disorganisation. The collagenous bundles in the immediate vicinity of the charred tissues become swollen and thickened, and large irregular spaces are formed by the sudden formation of steam in the tissues. The papillæ are swollen, thickened, and spherical, and become loosened from the epidermis. By the over-stretching of the horny layer of the skin and the sudden evolution of steam, the epidermis is raised, and bullæ form. Into these serum, sometimes mixed with blood, is effused. The blood-vessels and lymphatics of the tissues are seared and occluded. The zone surrounding the destroyed tissue is congested and swollen and shows considerable infiltration with leucocytes. The separation of eschars is effected by the growth of granulation tissue.

Treatment

Of the General Condition.—During the stage of collapse or shock the patient must be kept warm by means of hot bottles, warm blankets, or cotton-wool. Alcoholic stimulants, either by the mouth or as an enema, are clearly indicated, and should be given freely. Hypodermic injections of ether, strychnine, or brandy may be called for.

When pain is severe, morphia must be injected hypodermically.

As improvement takes place, hot drinks will do good if the patient can take them.

Local Treatment.—It cannot be too strongly urged that burns must be treated on the same rigidly antiseptic lines as other surgical wounds. In this connection it is important to note that Unna has invariably found pyogenic bacteria in the bullæ of burns.

The first step, therefore, is thorough purification by antiseptic agents, and that this may be satisfactorily accomplished a general anæsthetic is in most cases necessary. In the subsequent dressings of extensive burns, especially in children, the value of a general anæsthetic can scarcely be overestimated. The clothes are carefully removed, the burned area thoroughly, yet gently, washed with warm boracic lotion or a 1 in 100 solution of lysol. Blisters are opened and the raised epithelium removed; and a final wash with normal salt solution, to get rid of any excess of antiseptic, completes the preliminary purification.

The local application to be used depends largely upon the depth of the burn, its extent, and the time which has elapsed since its occurrence. In all cases the following indications have to be met—(1) the relief of pain; (2) the prevention of sepsis; and (3) the promotion of cicatrisation.

Local Treatment of Recent Burns of First, Second, Third, and Fourth Degrees.—*Picric Acid.*—Within recent years—thanks to Thiery and others of the French school—we have found in *picric acid* an agent which meets all our requirements in a way not previously attained by the older methods. It is employed in solutions varying in strength from 1 in 100, which is practically a saturated watery solution, to 1 in 50 when alcohol is added. It may be dissolved in sulphuric ether forming a solution of about 1 in 20, which is painted on to the affected area, and on the evaporation of the ether leaves a covering of a very fine powder of picric acid.

A useful lotion of moderate strength is made as follows:—

Take of Picric acid	1½ drachm.
Absolute alcohol . . .	3 ounces.
Distilled water to . . .	40 ounces.
Dissolve.	

After thorough purification of the burned area by antiseptic lotions, pads of lint or sterilised gauze are lightly wrung out of this solution, and applied over all the affected surface and for some distance beyond. A moderately thick layer of antiseptic wool is put on over this and retained in position by a bandage, preferably a many-tailed bandage, to obviate the necessity for much movement during dressings. A splint to ensure rest is advantageous when possible. It is important to observe that the dressing is applied *without* any waterproof covering—that is to say, it is not in the form of a poultice.

This dressing may be left in position for from three to seven days, according to the severity of the burn and the degree of asepticity. In burns of the first and second degrees it will be found that in three or four days all will be healed under a single application. In deeper burns, especially when the asepsis may not be absolute, it is well to change the dressing on the third or fourth day. Any portion of the original dressing which remains perfectly dry and adherent to the surface need not be removed, but should simply be moistened by pouring a stream of picric acid lotion over it. Any parts of the dressing which are moist from discharge must be removed, the surface thoroughly purified with boracic acid or lysol, and the picric acid pads reapplied. The dressing should be repeated once or twice a week according to circumstances.

Among the *advantages* of the picric acid method may be mentioned its simplicity and safety, as well as the infrequency with which dressings have to be changed. In a very few cases it has been found to cause temporary pain, but in the vast majority it is not only painless but even anodyne. Although not a powerful antiseptic it is sufficiently strong to maintain the asepticity of a burn which has been carefully purified at the outset. Its absolute dryness is an important factor in preventing the development of septic bacteria. But its most valuable feature as a dressing for burns is the marked power it has of promoting the proliferation of epithelium — its *keratoplastic action*—which seems to depend upon the coagulation of the albuminous exudate from the injured surface forming a non-irritating, aseptic protection to the young epithelial cells.

Its only *disadvantages* are that it temporarily stains the skin of the patient, and the hands of the surgeon and nurses. This to a large extent may be prevented by smearing the hands with vaseline before using it, and afterwards washing in methylated spirit or turpentine. Stains on cotton or linen clothes are readily removed by ordinary laundry processes, but remain permanent in woollen and flannel articles.

In a few isolated cases toxic symptoms—nausea, vomiting, diarrhœa, dark-coloured urine, yellowness of skin, and drowsiness—have been alleged to follow its use, but those who have had most experience with picric acid have not observed any symptoms attributable to its absorption. The writer, after an experience of over two hundred and fifty cases treated by this method, has not yet met with any untoward effects. In young children the solution may be diluted to about half its strength with advantage.

The *results* are best in superficial burns of the first, second, and third degrees, but the agent is useful as a primary dressing in burns of all degrees. After the sloughs have separated and a granulating surface is left, the usual applications for an aseptic healing sore should be substituted. When the area is large and cicatrisation slow, recourse should be had to skin-grafting by Thiersch's or one of the other methods available.

Ichthyol dnd Thiol.—These, as antiseptics and keratoplastics, are inferior to picric acid. They both cause considerable pain when first applied, but this soon passes off. Ichthyol is applied as a 30 per cent solution in water; thiol, either in watery solution 1 in 4, or as a powder, mixed with subnitrate of bismuth and a small quantity of iodoform.

Aseptic Treatment.—Mme. Nageotte has advocated the treatment of burns by simple aseptic dressings after thorough preliminary purification with chemical antiseptics. The results have not been entirely satisfactory, doubtless from the difficulty of obtaining absolute asepsis, and from the absence of any agent active in the promotion of epithelial regeneration.

Moist Applications; greasy substances, like carron oil, carbolic oil, and boracic ointment; substances calculated to occlude the burn, like collodion, dry powders, etc., are only mentioned to be condemned. They entirely fail to meet the indications for the rational treatment of burns on modern lines, and should be abandoned.

TREATMENT OF COMPLICATIONS.—Renal, pulmonary, cerebral, and other clinical complications of burns are treated on the same lines as similar conditions arising from other causes, and do not call for further mention here.

TREATMENT OF THE RESULTS OF BURNS.—*Ulcers.*—After the sloughs have separated from a burn and the stage of suppuration has been reached, an ulcerated surface is left which must be treated on the principles governing the management of ulcers. In the covering in of raw areas left by burns, skin-grafting finds one of its most useful applications. Means should be taken to prevent the production of deformities by the contracting scars during the healing of these ulcers.

Cicatrices.—The cicatrices following burns often constitute serious disfigurements, many of which may be amended by plastic surgery. They are also particularly prone to the various diseases of cicatrices (*q.v.*).

Deformities.—The contraction of extensive cicatrices, especially in the region of the face, neck, and flexures of joints, often lead to considerable deformity and interference with function, for which plastic operations may do good.

MEDICO-LEGAL ASPECTS

For medico-legal inquiries the following points may be noted:—

The Mode of Production.—Moist heat in the form of boiling water or steam leaves a widespread, red, soft, and sodden condition of the skin and underlying parts, without any actual destruction of tissues. Dry heat from molten metal, red-hot solids, resins, etc., results in a more limited damage of the tissues, which will be blackened, dry, and more or less completely destroyed by charring. The damage done by boiling oils resembles that from dry heat more than from moist, while flames produce injuries simulating both. The hairs of a part are singed by dry heat, but remain intact after the application of moist heat.

The Results of Explosions.—The injuries resulting from explosions of gas are of wide extent and superficial, and consist chiefly in scorching of the surface. In gunpowder explosions there are numerous small particles of carbon embedded in the skin.

Was the person alive or dead when burned?—In conflagrations persons are often suffocated by smoke, gases, or dust without being burned. In other cases they may be killed by suffocation and then burned. If there are vesicles on the skin, surrounded by a red and swollen area, and containing serous or sanious fluid, the presumption is that the person was alive when burned. The absence of these signs, however, does not negative the life of the person when subjected to the fire, as they often take some hours to appear, and death may have ensued before they had time to form. The skin under a vesicle produced during life is intensely injected, while that on a body burned after death is hard, dry, and yellow, and any blister which may have formed only contains gas. It is to be kept in mind, however, that heat applied to a dead body while still warm produces appearances closely resembling those of burns inflicted during life. The main difference is that the fluid in the vesicles in the former case is thin and watery, and contains a very small proportion of albumen.

In burns produced by red-hot solids during life the skin round the destroyed tissue is usually white, with a deep red ring beyond it. In a dead body this red area is absent, and the whole of the destroyed surface is deadly white.

How long did the person survive the burning?—This question will be answered by noting whether there are present only the signs of actual burning, or whether advanced inflammatory signs have supervened. Suppuration, the separation of sloughs, the formation of granulation tissue, and the onset of gangrene indicate a considerable lapse of time.

Marks of Violence on a Burned Body.—The body may have been burned to conceal marks of violence. Careful examination may reveal such marks, unless the destruction be too complete.

Spontaneous Combustion.—It is almost needless to say that the belief that spontaneous combustion may take place in the human body can no longer be maintained.

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Bursæ, Injuries and Diseases of.

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INTRODUCTION.—Two varieties of Bursæ are found, the true and the false. A true bursa is constant, while a false bursa appears adventitiously over some abnormal prominence of bone, *e.g.* the so-called bunion, or the thickening over the prominent spinous processes in an angular deformity of the spine. The size of a false bursa is a fair indication of the duration of the abnormal prominence of bone, and the longer the false bursa has existed, the nearer does it approach in structure to the true bursa. A true bursa is a simple synovial sac placed between two surfaces to prevent friction, and such sacs are either deep-seated or subcutaneous.

In structure a bursa is a sac consisting externally of areolar tissue of varying density, and lined internally by a more or less perfect synovial membrane of flattened cells. The cells are not continuous over the whole surface of the synovial membrane, but are distributed in patches, the spaces between being filled by the ground substance of the connective tissue of the wall. Numerous blood-vessels and some nerves are found in the walls, and when enlargement takes place the vessels increase in size and number. The cavity of a bursa is a lymphatic space in the same sense as is the synovial cavity of a joint, in fact, a bursal cavity is an isolated articular cavity. The false bursæ are at first recesses in the subcutaneous connective tissue, and are not bounded by true synovial membrane; but after a time patches of cells which have an epithelioid appearance are seen in the walls, and a more or less perfect bursal cavity is formed. The synovial sheaths of tendons are true bursæ, and facilitate the movements of the tendons in their osseo-fibrous grooves. These sheaths in no way differ in structure from bursæ.

SITUATIONS OF BURSÆ.—In the *head, neck, and trunk.* Apart from the intra-articular synovial sacs of the temporo-maxillary, vertebral, and intercostal articulations, bursæ are here very few in number. An important one is interposed between the posterior surface of the body of the hyoid bone and the thyro-hyoid ligament. It sometimes becomes enlarged and forms an encysted fluid swelling, projecting in the floor of the mouth.

In the *upper extremity* bursæ are very numerous. An important one surgically is the large subscapular bursa between the subscapularis and the deeper muscles. This bursa is often the site of the dry form of bursitis, when movements of the scapula become painful, limited, and accompanied by distinct creaking. If fluid is poured out the scapula is displaced backwards. A large subcutaneous and often false bursa is found over the acromion process of the scapula, especially in those who carry weights on their shoulders. In the immediate neighbourhood of the shoulder-joint are three bursæ, one beneath the tendon of the subscapularis, which frequently communicates with the joint, one beneath the tendon of the infra-spinatus, which rarely opens into the joint, and one large sac beneath the deltoid, which has no direct connection with the joint. The investing sheath of the long head of the biceps may be regarded as a bursa. Between the tendon of the teres minor and the shoulder a bursa is occasionally found, and one between the tendons of the teres major and latissimus dorsi. In the neighbourhood of the elbow-joint are three well-known bursæ, one between the skin and olecranon process, a second between the tendon of the triceps and the upper part of the olecranon, and a third intervening between the biceps tendon and the tuberosity of the radius. At the wrist the tendons of both the superficial and deep flexors, as well as the median nerve, are surrounded beneath the annular ligaments by a large loose synovial membrane which extends upwards to the radio-carpal articulation and downwards to a little beyond the bases of the metacarpal bones, being prolonged further down in the tendons of the little finger than in the others. A sheath exists around the tendon of the flexor longus pollicis as it passes beneath the annular ligament and communicates with the large flexor bursa at the upper border of the annular ligament. The large bursa of the palm of the hand is important as the site of the so-called "compound" ganglion or tubercular form of bursitis. Beneath the insertions of the following muscles bursæ are found—extensor carpi radialis longior and brevior, also between the surface of the former muscle and the supinator brevis. False bursæ also form over the knuckles and interphalangeal joints in those who labour manually.

In the *pelvis* and *lower extremity* the bursa over the tuber ischii is large and multilocular and frequently enlarges in tailors and weavers, hence originates the expression "weavers' bottom." Beneath the gluteus maximus are two bursæ, one multilocular between it and the great trochanter, and one between the muscles and the vastus externus. Other bursæ are found beneath the insertions of the gluteus medius and minimus. While between the obturator internus and the margin of the small sciatic foramen is a bursa which is often continuous with another between the tendon of the muscle and the hip-joint. At the front of the joint is a bursa beneath the psoas which often communicates with the joint. Above the knee there is a bursa beneath the quadriceps extensor also in communication with the joint. The bursæ in relation with the patella and its ligament are three in number. In front of the lower half of the patella and upper half of the ligament is the pre-patellar bursa of "housemaid's knee." Over the lower half of the ligament is a second bursa, and between the ligamentum patellæ and the tubercle is a third bursa. In the popliteal space are the following bursæ: On the outer side and above the joint are found one beneath the outer head of the gastrocnemius, and one beneath the tendon of the popliteus, which is almost always an extension from the first. On the outer side below the joint are bursæ between the tendons of the popliteus and biceps and the external lateral ligament. On the inner side there is a bursæ between the inner head of the gastrocnemius and the femur, which is prolonged between that muscle and the semi-membranosus, and often communicates with the joint. There is also a bursa between the semi-membranosus and the head of the tibia which is frequently enlarged, and sometimes a bursa between the tendons of the semi-membranosus and semi-tendinosus. A bursa also separates the tendons of the three inner ham-strings from the internal lateral ligament, and is prolonged beneath the insertion of the sartorius. In the foot beneath the insertions of the tibialis anticus and sometimes of the peroneus brevis a bursa is found. One important bursa is found between the upper part of the posterior surfaces of the os calcis and the tendo Achillis. It is frequently inflamed, and the affection so constituted is called "Achillodynia."

INJURIES OF BURSÆ.—These structures, from their exposed situations, are particularly liable to traumata either of accidental and violent character, or of a frequently recurring nature, and from the latter originates the condition known as chronic bursitis. A blow in the neighbourhood of a bursa results either in a contusion around the bursa or hæmorrhage into the bursal cavity from rupture of the vessels supplying it, an event especially liable to occur in a hæmophilic patient, or the bursa is ruptured, a very probable event if it already contains fluid, or it may be cleanly torn open or lacerated. Injuries not sufficient to cause severe hæmorrhage are followed by acute serous effusion. But if hæmorrhage occur it may either be absorbed or sometimes followed by suppuration, or failing to be absorbed it results in a fibroid bursa. Blood may be effused in such large quantities as to mask a severe injury of the bone beneath, *e.g.* fracture of the patella or of the olecranon.

The treatment is as follows:—If there is no abrasion of the surface a cooling application should be employed for the first thirty-six hours and the part kept at rest. Then hot applications may be used to assist absorption, assisted by pressure and gentle friction. Should the part become acutely inflamed, it is better to make a free incision, for the bursal sac is a lymph space and absorption from it readily occurs. If the bursa is wounded or lacerated it should be thoroughly cleansed with antiseptic fluids, and a gauze drain placed in the cavity for two days.

BURSITIS.—(*a*) *The Simple Acute Form* (*Acute Hygroma*) is etiologically of two varieties, Traumatic or Primary and Secondary from extension of inflammation in the neighbourhood. Traumatic hygroma follows injuries, especially contusions and penetrating wounds; if it occur in bursæ already inflamed, suppuration often ensues, and hygroma is occasionally seen from excessive over-use. The secondary form is due to extension to the bursal sac of inflammation in the vicinity, although the sac may not be in direct communication with the part. Acute secondary bursitis is therefore seen in connection with a boil, carbuncle, erysipelas, suppurative arthritis. The secondary form is either serous or suppurative, and the latter is either localised or diffuse. The serous form (acute serous hygroma) is accompanied by phenomena of gravity such as very acute pain, redness, œdema, and the formation of a tumour often within the first twenty hours. If the effused fluid remains serous, the swelling lessens in five or six days, and the inflammatory symptoms subside, and the hygroma either disappears or becomes chronic, or pus may form. Should the suppuration be localised to the bursa, it will, if left to itself, burst, either externally and be followed by the formation of two or three fistulous openings, after which the bursa may shrink and disappear; or it will invade the neighbouring tissues and cause extensive cellulitis, and we have seen a suppurating bursa patellæ followed by necrosis of that bone and by acute arthritis. The treatment is evidently to open the inflamed bursa freely so soon as any suspicion of pus arises.

(*b*) *Simple Chronic Bursitis* (*Chronic Hygroma*). — *Etiology.* — It frequently follows acute but more often is the result of occupation, as in housemaids, cobblers, tailors and miners. Verneuil describes bursæ as existing over tumours, and Cruveilhier met with a large serous bursa between a mammary scirrhus and the pectoralis major. Pre- and peri-hernial cysts are often bursal in their nature. False bursæ are met with over the prominences in clubfoot and in bunion. The varieties of chronic hygromata are the cystic, proliferating, fibrous and hæmorrhagic. A cystic hygroma is the most frequent, and corresponds pathologically to hydrarthrosis. It is sometimes bi-lobed in shape, but is usually unilocular rarely multilocular. The wall is composed of thickened fibrous tissue, cartilaginous in places, or incrusted with calcareous plates, and the bursa often lies in a fatty envelope, *cf. hygroma lipomateux de la nuque* in porters. The contents are either clear or opaque fluid, of yellowish colour, varying in consistence and containing many cholesterine crystals. Sometimes, however, it is bloody or purulent. In the wall there are often large arteries present, which may give rise to hæmorrhage. The proliferating hygroma is generally unilocular. The cavity contains a variable quantity of liquid, and has loose bodies floating in it which have been detached from the interior of the sac. On the latter are seen numerous vegetation-like bodies which are wart-like in appearance, sessile or pedunculated, and composed of fibrous tissue infiltrated with cartilage, and sometimes with uric acid or phosphate of lime. The fibrous hygroma is met with, especially in front of the knee, as a firm indurated mass, in the centre of which is found a little fluid. The hæmorrhagic hygroma is either the result of traumatism, or the blood may be spontaneously effused. The fluid in the cavity is thick and black, or may consist almost entirely of clotted blood. Volkmann has described a parallel condition in joints as "pachysynovitis hæmorrhagica."

The symptoms of chronic bursitis are not difficult of recognition. In an appropriate situation a small rounded or flattened tumour commences which is indolent, nearly painless, fluctuating, and on pressure may be rolled under the hand, giving a feeling of fine crepitation. A fibrous hygroma is from the first hard and unyielding in character, while a hæmorrhagic hygroma has very few distinguishing characteristics. The course of a hygroma is chronic. It may disappear entirely, or it may undergo progressive sclerosis and contraction of its walls. The chronically enlarged bursa is exposed to rupture, hæmorrhagic effusion, and suppuration. If it be subcutaneously ruptured the fluid is absorbed by the tissues, but the bursa reappears.

Treatment.—There are practically two methods of treatment—obliteration and excision of the sac. In the early stages of chronic bursitis, if the irritating cause be removed, the bursa will often subside, only to reappear when the cause is at work again. Counter-irritants are sometimes of temporary service. The means adopted for obliteration of the sac are incision, and the application of nearly pure carbolic acid, or simple incision and stuffing the cavity with gauze, so permitting it to granulate up entirely. But, in all forms of chronic bursitis of long standing, especially the proliferating and fibrous, complete removal of the sac is necessary.

Gouty, Gonorrhœal, and Rheumatic Bursitis.—Allusion has already been made to the deposit of urate of soda in the walls of bursæ, and if the subject be affected with well-marked gout, this salt is often deposited continuously over the whole interior of the sac and in its walls. The bursæ most frequently affected are the olecranon, patellar, and the subcutaneous bursæ of the hands and feet. In gonorrhœa and acute rheumatism the bursæ in the neighbourhood of an inflamed joint often sympathise acutely. In the early stages a dry hygroma is present, which is painful and finely crepitating to the touch, but serous effusion sets in to a varying degree, and the bursa swells somewhat. Its symptoms then approach those of the serous variety of acute bursitis. The diagnosis is made and treatment carried out by recognising the associated conditions.

Tuberculous Bursitis.—This was formerly thought to be rare, but many cases which were published under the title of fungous disease of the bursæ are now recognised as tuberculous bursitis. The sites of the affection are the olecranon, sub-deltoïd, sub-ischiatic, pre- and infra-patellar, malleolar, psoas and popliteal bursæ.

Two varieties of tuberculous bursitis are met with, the primary and the secondary. Of the existence of the former there can be no doubt, and all conditions are present in bursæ for the development of tubercle in suitable subjects, such conditions being—exposure to injury, abundant fibrous tissue, and the peculiar synovial character of the lining wall of the sac. Secondary bursitis is distinguished from primary by its being associated with tubercle of the neighbouring joint. It is not essential that there should be any direct communication between the synovial membranes of the bursa and joint. Pathologically three forms are recognisable—fungous hygroma, hygroma with rice-grain bodies, and myxomatous or mucoid hygroma. Of these the fungous hygroma is most common in type. At first the cyst is simply serous and its wall is not thickened, but it soon becomes covered by small tubercles. This stage often escapes observation, but later granulation tissue forms and caseates, and pus is seen, so that the general appearance of the cavity is like that of tuberculous synovitis. It is probably a slow form of tubercle which affects bursæ, as the results of inoculation of animals take a long time to appear. In the rice-grain bodied form of hygroma there is but little fluid. A large number of these curious bodies is present, and that they are truly tuberculous is shown by the results of inoculation (Nicaise, *Revue de chir.* 1885). This form of tuberculous bursitis is frequently met with in the large palmar bursa, and used to be known as "compound ganglion." The mucoid form of tuberculous hygroma has been described by Critzmann (*Médécine moderne,* 1890, p. 638). There is found a cyst filled with gelatiniform material, translucent and red or yellow in colour. The nature of this material has been conclusively proved by inoculation. The symptoms are those of a slow chronic swelling, often thick-walled and giving the impression of a solid bursa as the fluctuation is obscured. In the rice-grain form fine crepitations are present.

Unlike other bursæ, the more tuberculous a bursa grows, the softer and more semi-fluctuating does it become. This is a diagnostic sign of great importance. Eventually a fistula forms, but before that has occurred, extension has taken place to bones and joints. The only permissible treatment is extirpation.

Syphilitic Bursitis.—It is met with in the secondary stage of syphilis, and as the subacute serous form, and in the tertiary stage as the fibroid variety which becomes gummatous. The latter variety may occur as long as twenty years after the primary infection, and frequently follows an injury. The seat of election is the patellar bursa. The progress of a tertiary syphilitic bursa is very slow, but ultimately the tumour becomes adherent to bone or skin and ulcerates. The diagnosis is often difficult, as it may be mistaken for simple chronic hygroma, old hæmatoma or nodes, and when it ulcerates it may simulate tubercle. It is, however, very indolent and slow in progress, but when small readily yields to treatment. If of large size or ulcerating it should be completely removed by excision.

Neoplasms.—The innocent forms met with are fibro-enchondroma and myxoma. The latter has been seen by Chevasse three times. In one case the tumour had attained the size of a child's head and was freely ulcerated on the surface. Fibro-enchondroma is comparatively rare. Considerable difficulty is met with in diagnosing these conditions, and the removal is frequently undertaken, not because their nature has been recognised, but because of their size. Malignant new growths are either sarcomatous or endotheliomatous. The sarcoma is either of the soft or round-celled type, or is of the rapidly breaking-down hæmorrhagic variety. With reference to endothelioma, Döllinger has met with one case in a fistulous bursa of a woman aged 58, and Mickulicz has seen a similar case. The softer varieties of neoplasm may attain an enormous size, and Ranke (*Archiv für klin. Chir.* 1886) has met with one 62c/m in circumference. The treatment of these neoplasms is complete removal.

LITERATURE.—1. Duplay et Reches. *Traité de chirurgie.* 2me edition, 1897, article "Bourses sereuses."—2. Critzmann. "Hygroma tuberculeux á type myxomateux," *Médécine moderne,* 1890, p. 638.—3. Nicaise. "Nature tuberculeuse des hygromas," *Revue de chir.* 1885.—4. Volkmann. "Die Krankheiten der Schleimbeuteln," *Handbuch der Allgemeneine chir.* 1882.

Cachexia.—This term, derived from the Greek words κακὸς and ἕξις, and meaning literally a bad habit of body, is one which has been used from the very beginnings of medicine. It never has had, however, a very precise meaning, and the following is its definition as given by Copeland:—"Depravity of the constitution, without fever, affecting more or less the solids, the circulating fluids, and the secretions." In this way cachexia is to be regarded as meaning the outward and mainly the facial expression and characteristics resulting from the profound and complex changes in nutrition induced by a malady of some chronicity. It is to be noticed that cachexia is to be distinguished from diathesis, by which is meant a constitutional disposition in virtue of which an individual is liable to certain local affections of the same nature. It is also to be distinguished from the malady acquired as the result of some innate weakness or unhealthy surroundings, and no less from the conjoined symptoms and signs of the disease induced by such.

With the diathesis, with the conjoined symptoms, and with the malady itself, it has, however, frequently been confounded, and its distinction as a term from marasmus, dyscrasia, and many others, is not well marked.

There is no doubt that as our knowledge of healthy and diseased processes is becoming more exact, the term cachexia is becoming less and less made use of. In olden times the cachexias and the fevers shared a great part of the physician's attention, whilst now, the term, if mentioned in a text-book, is seldom deemed of sufficient importance to merit a place in the index. For this disuse our more precise knowledge of disease, and our clearer grasp of the association of symptoms, form, as has been said, the explanation. The terms cachectic angina and cachectic liver abscess, of a previous generation, are hardly ever used now, the sore throat and liver abscess being ascribed to infective organisms acting on an individual of lowered nutritive power. Further, the scorbutic cachexia, the syphilitic cachexia, the cardiac cachexia of the French writers, and many others, are recognised now rather as a more or less definite association of certain symptoms or signs. It follows, therefore, that although in reality every disease has its cachexia, yet the cachexias which require detailed consideration at the present time, are only three in number, viz. the cancerous, the malarial, and the metallic.

Cancerous Cachexia.—By this is indicated the facial aspect and the general appearance of feebleness and loss of flesh met with in individuals who have been for some time suffering from cancerous disease. In its marked forms the face is pale with a yellow or greenish-yellow tint, the cheeks hollow, the nose pointed, and the eyes sunken. Corresponding with the emaciation and muscular weakness the movements of such patients are slow, and as the result of the more or less constant pain, and of the general organic sensation of unwellness and feebleness, the expression is sad and gloomy and betokens suffering. The colour of the skin in such cases is due to changes in the blood and skin pigment, and these in their turn are probably brought about by the resorption into the blood of the fluid secretion of the cancer cells. It is to be remembered, on the one hand, that the cancerous cachexia may be closely simulated by other morbid conditions, and on the other, that cancer may be present and yet cause no cachexia. This latter might be explained on the ground that little or no pain is present, and that no resorption is taking place from the diseased mass. It would seem that the cachexia is usually most evident in cases of ulcerating cancer and in cancers affecting the stomach, intestine, and tongue.

Malarial Cachexia.—By this is meant the changes which are apt to supervene in an individual who has suffered from one or more attacks of malarial fever, or, but to a less extent, who has been for long times exposed to malarial poison. It is associated specially with changes in the blood and spleen, but the liver, kidney, and nervous system are apt also to show morbid changes. The cachexia shows itself principally in the colour of the skin. It is pale, resembling in this respect ordinary anæmia, but it often reveals a yellow aspect, which has been compared to that of old wax, or a brownish colour, which has been compared to that of ginger-bread. With this the affected individual usually presents an aspect of marked feebleness, and shows a proneness to dropsy of the dependent parts.

Metallic Cachexias.—Of these the lead and the mercurial are the examples which have received most attention. The lead is the only one which merits here detailed reference. This cachexia, which rapidly manifests itself in individuals who have been exposed to the causes of lead poisoning, consists mainly in an alteration in the colour of the skin. This in its pallor resembles anæmia, but differs from it in presenting, along with the pallor, an appearance of what is best expressed by dirtiness, the result of the presence in the pale skin of minute particles of lead. In addition, the blue line along the softened gums, the fœtor of the breath, and the peculiar paralysis, etc., render its recognition easy.

Cæcum. *See* Appendix, Diseases of.

Cæsarean Section. *See* Obstetric Operations.

Caisson Disease. *See* Spinal Cord.

Cancer. *See* Tumours.

Cancrum Oris. *See* Stomatitis.

Capillaries, Diseases of.

Congenital Angioma—
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The most common and important affection involving capillaries is angioma. This may occur in any part of the body, but is most frequent in the skin and subcutaneous texture, so that we may confine our description to that region. Probably the most elastic and comprehensive classification of the angiomata is the anatomical.

Angioma:
Capillary = Telangiectasis; Erectile tumour; Cavernous tumour.
Venous = Phlebectasis.
Arterial = Cirsoid aneurysm.
Lymphatic = Lymphangioma.

Capillary angioma is best considered under two heads: the congenital and the non-congenital.

I. Congenital Angioma. Nævus—may be subdivided pathologically or clinically. In the one view we have the simple, venous, and arterial; in the other the cutaneous, subcutaneous, and mixed. We shall presently see the value of these distinctions.

Histology.—On section the simple nævus presents the appearance of a finely reticulated sponge with imperfectly marked lobulation. The laminæ are thin and fibrous, and lined by epithelium continuous with that of the blood-vessels. It may or may not be surrounded by a capsule. A nævus grows in two ways—intrinsic or extrinsic. If its growth is intrinsic, and especially if it be slow, it pushes parts aside and forms a capsule. But a nævus often invades its surroundings like an inflammation or a malignant tumour. What is the influence which metamorphosed capillaries can thus exercise on their neighbours it is difficult to conceive. But I have seen one grow by invasion in a week or two from the size of a shilling till it occupied half a baby's face and head, occluded its eye, and deformed its ear. In such cases, and in so far as a nævus may occupy the skin, there is no capsule, a point of importance in connection with treatment.

In the vascular arrangements there are many modifications. Thus it is not unusual for a nævus to pulsate from the beginning, when tortuous and dilated arterioles may be dissected out, although there is certainly no form which in the least resembles histologically the cirsoid aneurysm, or even perhaps the aneurysm by anastomosis of the adult. Similarly enlargement of venous radicles may enter largely into the composition of a nævus—so largely, indeed, as greatly to mask its original capillary character. If you put on the stretch skin which is occupied by a small and recent nævus, you will display a most beautiful network of visible capillaries often in parallel rows, with perhaps a winding arteriole or dilated vein. From this simple dilatation, and it may be multiplication of capillaries, presumably arise by pressure on the intervascular structures and formation of fresh intercommunications the appearances already described.

Clinical History.—There may be seen a slight stain at birth, and as a rule a nævus declares itself if at all within a fortnight. It is very variable in its rate of growth. Its most rapid rate is generally early. After the first few months it tends to grow with the child. There is a great tendency to spontaneous disappearance. More than half of the subcutaneous and mixed varieties are naturally cured. The cutaneous variety, the port-wine stain, is, however, prone to persist through life. The subcutaneous and mixed varieties are likely to persist in proportion as they are associated with or acquire arterial, venous, or degenerative changes.

The natural cure is brought about by a fibrous atrophy probably inflammatory in character, and certainly capable of being excited by inflammatory causes to which a nævus is very sensitive. This process may be modified in various ways. In the atrophy portions of the tumour may be blocked off and form cysts. The altered blood in them is more thin and watery, and the deposit of fibrin on their walls less copious than in hæmatocele or extravasation cysts. I am not sure that these cysts are always blocked off blood-vessels, because I have found them purely serous, even among others containing altered blood. But probably the most frequent degeneration is fibrous. The nævus is cured, but involution of the fibrous tissue fails. I have not met with a sarcomatous change in nævus unconnected with the pigmented mole.

Diagnosis.—The symptoms need only be considered in so far as they aid diagnosis. The mixed variety of nævus may be mistaken for meningocele and spina bifida, the subcutaneous or degenerated for fatty, cystic, and sarcomatous tumours.

The most important diagnostic difficulty is with meningocele, which I have several times had sent me for nævus, from their similarity in appearance, consistence, and compressibility. The mere fact that the growth occurs in the middle line should put one on guard. In that line only lies the difficulty. The meningocele is seen nowhere else. Cohesion to the bone calls for additional care. A nævus may cohere, but if on compression a neck-like connection, and still better if the contour of an orifice in the skull can be felt, a very important distinction is made out. If it be not absolutely congenital it is nævus. If an introduced needle be felt free in a cavity, and clear fluid with cerebral characters be removed, no doubt remains. The same considerations apply to spina bifida, which is also sometimes mistaken.

The sarcoma as it occurs in infants is apt to be very vascular. and looks like a nævus. The congenital, or nearly congenital origin, the shape, compressibility, consistence, and situation serve to distinguish a nævus from it and other tumours.

Treatment.—No nævus in a child should be submitted to treatment unless it be clearly increasing, or likely to do harm from its situation or condition. Many nævi disappear, and Nature's result is as good as any. The reason for treatment is not usually danger, but disfigurement. The danger comes from wound or ulceration. It is therefore slight. If the tumour be stationary, not ulcerated, and not doing harm, it is best to wait. I leave out of account harmless placebos such as collodion or gentle pressure.

The methods of treatment are very numerous.

They may be arranged as—

1. External escharotics or irritants; 2. Subcutaneous escharotics or irritants; 3. Ligature; 4. Excision.

1. There are three conditions under which this form of treatment should be adopted—

(*a*) A tiny nævus, with radiating vessels, is common on the nose or cheek. It generally goes away, but sometimes increases. It may be so easily destroyed by a needle with nitric acid, by a heated point, or the negative electrolysis needle, that it ought to be cured without delay.

(*b*) Flat nævi of the skin on the covered parts of the body may be well treated by Richardson's sodium ethylate. It leaves little scarring.

(*c*) Our only method of treating the port-wine stain of the face without leaving a worse mark is by the long-continued application of irritants. Squire's knife, multiple puncture, and all other means have with me been very disappointing. Cure by means of an irritative dermatitis is not certain, and requires much patience and perseverance; but it does no harm, and is sometimes successful. I think strong iodine is the most manageable irritant.

2. I have entirely given up the subcutaneous injection of such substances as tannin, perchloride of iron, and carbolic acid, as well as the subcutaneous use of the heated wire, in favour of electrolysis. All of them act in the same way, viz. by the destruction of texture. But by electrolysis the destroying agents are introduced in infinitesimal division, and therefore are much more under the surgeon's control than by any other method. Moreover, the danger of subcutaneous injection without a temporary ligature has been frequently demonstrated by the sudden death of the patient. There are one or two rare cases in which the platinum wire heated by electricity subcutaneously might be used, but almost invariably for subcutaneous use electrolysis is the best agent.

The mode of using it and its value will be considered under the article "Electrolysis." Meantime it suffices to say that for mixed and subcutaneous nævi on exposed parts of the body, and for nævi elsewhere, which are very large or otherwise inoperable, electrolysis is safe and certain although slow.

Ligature.—I think there is scarcely any case in which this method is now required.

Excision.—The advantages of excision over electrolysis are its greater certainty and rapidity. The former is slight because excision may fail, and electrolysis rarely does so. The latter is undoubted and considerable. Excision requires only one operation, electrolysis usually more. In cases, therefore, where a scar is of no importance, and the size and relations of the tumour are not such as to make the operation dangerous, excision is preferable to any other method.

II. NON-CONENTIGAL CAPILLARY ANGIOMA is in the simple form rare, most usual in internal organs, of exceedingly slow growth, but with little tendency to retrogression. More common are the venous and arterial.

These three varieties correspond to the varieties of nævus histologically. They present, as above seen, differences from nævus clinically, and to the last belongs that rare affection called osteo-aneurysm. A fourth variety has no analogy among congenital varieties, and to it the name aneurysm by anastomosis may properly be given. It consists of metamorphosed capillaries in which large cavities are connected with dilated arteries on the one hand and dilated veins on the other. Many of the characters of arterio-venous aneurysm are here to be seen mingled with others more proper to a capillary or venous tumour.

In treatment we are here also restricted to electrolysis and excision. The former most certainly should be tried first, as it is practically without risk, and holds out a fair prospect of success. These varieties, and especially the aneurysm by anastomosis, are, however, much more resistant than congenital forms. When electrolysis fails the propriety of attempting excision must be determined in each case by the risk. It is always to be remembered that these tumours have little tendency to shorten life, and that only the trouble they give, chiefly by disfigurement, pulsation, and murmur, warrants interference. Operation by excision is always serious, often impossible.

The consideration of the other forms of angioma limited in origin to the arteries, to the veins, and to the lymphatics, will be found in the articles on these subjects and on "Tumours."

Carbolic Acid. *See* Toxicology.

Carbonic Acid. *See* Toxicology, and Air, Examination of.

Carbuncle. *See* Boils.

Carpo-pedal Contractions. *See* Tetany.

Catalepsy.

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Definition.—Catalepsy is a nervous affection characterised by a plastic rigidity of voluntary muscles; by unconsciousness, including the abeyance of common and special sensibility; and by the abeyance of reflex irritability.

Etiology.—Females are disproportionately predisposed to catalepsy, and the period of adolescence is a common age for its occurrence. The affection is predisposed to by any conditions which make for neurasthenia; it has obvious katamenial relations in many cases; and, both in males and females, there are usually some suggestions of hysteria. Very commonly the first attack is occasioned by some mental or nervous incident of a violent nature—a fire, an assault, great religious excitement, a fall, a sunstroke, a sudden affliction, and similar emotional or nervous shocks. Cataleptiform affections may appear in association with that form of insanity called stupor, with hysteria, with tetanus, with chorea, and with epilepsy, so as to suggest an etiological relationship.

Symptoms.—The first attack of catalepsy may be quite unexpected. But the affection is apt to be paroxysmal and even periodic. Even in such cases the patient may be quite well between times, but it is very commonly the case that a more or less sudden giddiness, or headache, neuralgia, hiccough, or visual disorders immediately precede a seizure. Therein we observe a first resemblance to epilepsy, and, to appreciate the condition, we must constantly keep that comparison in mind.

Whether there has been warning of some minutes or longer, or if there has been none, the patient is, at the instant, usually aware that she is about to fall. With perhaps some vague efforts to kneel or to reach a seat, she sinks helplessly to the floor or subsides in her chair. Apparently, in a typical seizure, the consciousness of the patient is as it may be in a deep swoon. The last fact that she is aware of is that she has lost control of her muscles, and she appears to know no more until she recovers some minutes or hours or even days later. She has then no recollection of what has transpired meantime. Whether or not there be any isolated mental activity during a seizure, we have no means of knowing.

Meanwhile motor symptoms of a most interesting character have been developed. There is no violent tonic spasm in catalepsy. But there is fixation of the muscles of a nature which is curious and not easy of explanation. An epileptic will crash to the ground as a rule, and then, of course, clonic spasms follow. A hysterical patient will fall with some slight arrangement of her limbs and skirts, and other signs of apparent purpose in her movements or in her pose. But in a typical cataleptic seizure the patient drops off as in syncope, and her muscles become fixed in the first attitude at which she comes to rest. If you try to lift a limb, or rearrange the body generally, you will find that the muscles have stiffened as if in rigor mortis. Then by degrees they gently relax, so as to adapt the pose to the action of gravitation, and the trunk and head and limbs all sink flatter on the ground or couch. At this stage the muscles have reached the characteristic condition of plastic rigidity, the distinctive feature of catalepsy known as *Flexibilitas cerea.* The figure is not now cast in metal but in wax, and is plastic under your fingers. It retains any pose which you make it assume. There is no automatic movement of a purposive kind, no lifting of an arm or hand or foot, and there are no spasms. If you raise a limb to a position of strain, the muscles will retain that pose for a time, and then by slow degrees will allow the limb to sink to a position determined by gravity.

At this stage the sensibility of the patient will be abolished. The skin is apparently quite insensitive to touch, to heat, to pain, to electricity. Special sensibility is abolished. Reflex irritability is also in abeyance. Even the conjunctival reflex is probably abolished. Respiration is very shallow; cardiac action is slight; and the temperature slowly falls.

A typical seizure has been described. Very commonly there will be important modifications in the symptoms. In particular the consciousness and the sensibility of the patient may not be abolished, though powerlessness and plastic rigidity may have supervened. The reflexes may not be greatly impaired, though common and special sensibility is in abeyance. Hypersensibility has been reported as a rare variation. As in other such diseases, there are cases of spurious catalepsy, in some of which symptoms of hysteria are conspicuous, and cataleptoid elements are frequently prominent in allied affections.

Treatment.—*During an attack* treat the patient as for an epileptic seizure. Put the body in a position of ease, and do not disturb the patient.

In the majority of cases spontaneous awakening occurs at the most after a few hours. It is difficult to see what good is served by violent attempts to arouse patients from coma. Gentle means may occasionally be tried—a sharp puff of wind on the face, a breath of smelling salts, a pinch of snuff, or even a very mild splashing of cold water. Avoid emetics and painful shocks of electricity. If the attack continues, food may be necessary. If so, pass it into the stomach by the tube, or, in the first instance, empty the rectum and administer a nutrient enema. Draw off the urine if there are reasons for supposing that there is any considerable accumulation. If the patient is comfortably in bed and these measures are adopted, there is no reason for supposing that the cataleptic sleep will be hurtful, even if it is protracted over several days.

After an attack, treat the patient as for neurasthenia. Avoid regarding the case as hysterical. Consider it rather as epileptoid. Try to discover any mental or physical source of irritation, and remedy it if possible. As in chorea, the patient may be greatly benefited by a change of scene, especially if that entails a removal from the chief source of annoyance or excitement. Special regard should be had to pelvic factors in the disease.

PROGNOSIS.—The prognosis in each seizure is good, and the prospect of immunity from attacks is also good, and increasingly so according to the age of the patient.

Differential Diagnosis.—In *stupor* there is more or less persistent mental disorder. In *hysteria* the plasticity of the limb receives some added effort of its own. A cataleptic limb is moved from one position to another, and will retain the position in which it is placed for some time. A hysterical limb generally moves. By judicious change of pressure or of direction you may observe the hysterical limb continue to move without your aid, or continue to oppose a strain which you have withdrawn. In *tetanus* there is spasm, and generally obvious signs of pain. In *epilepsy* there are first spasms, and in the later stages the plastic rigidity is absent. In the cataleptic state induced by *hypnotism* the symptoms, in this country, vary considerably, and in general there is, as in hysteria, a feeling on manipulating the limbs as if the patient were following your movements. In *death* physical signs discoverable by auscultation are wanting, and the *rigor mortis* is not plastic.

NATURE AND PATHOLOGY.—We have no definite knowledge of the pathology of catalepsy. The nervous condition previous to a seizure must be held to explain, in a histologist's sense, the nature of the lesion, and it probably is comparable to other related diseases—mania, hysteria, epileptoid states, etc. As to the seizure itself, and its effects, it is important to consider what are the mechanisms which are chiefly involved.

It is obvious that the lesion is partly cortical, and that it involves mechanisms which subserve perception and volition. The coma seems to have features which distinguish it from that of sleep, of induced hypnosis, of epilepsy, or of swoon. The mode of invasion is characteristic, and the muscular accompaniments, and the depth of insensibility. Apparently the lesion is one which specially involves the outward realm of mental mechanisms—those which subserve muscular activities. The loss of voluntary movement is the prelude to the curious phenomenon which persists—the plastic rigidity in voluntary muscles. The invasion of consciousness would seem to be somewhat similar to what occurs in certain epileptiform attacks (not epileptic) in which the spasms of voluntary muscles initiate an attack in which consciousness is thereafter lost. In catalepsy there is paralysis of voluntary movement, followed by paralysis of consciousness. The most interesting phenomenon, however, still remains—the balanced and coordinate muscular contractions which determine rigidity of the limbs, and their persistent sensibility to pressures which determines their plasticity. That phenomenon undoubtedly suggests, as Gowers has pointed out, an affection of the muscle-sense organs.

Afferent fibres from the muscles, fascias, and joints, and corresponding motor tracks, complete a reflex arc whose apex is in the spinal cord. These mechanisms subserve the function of unconscious balance and pose. In normal life, however, that reflex arc has an extension upwards to the cortex, which makes possible a voluntary control of its activities. In catalepsy the higher activity is in abeyance, and the lower mechanisms are active in excess. The rigidity then is simply a secondary, positive sign of the removal of conscious control and the plasticity of it is part of its normal function—to adapt pose to stress. But the extraordinary thing is that this function *should* persist when others have been abolished—an isolated and excessive activity in a general condition of profound coma. We are therefore led to conjecture that there is something very like an epileptiform seizure in capalepsy, and that the motor convulsion, if there is one, is a convulsion in this mechanism to which we have referred. The absence of obvious spasms, either tonic or clonic, signifies only that the convulsion is not in a mechanism of large movements, which we already know. The mechanism referred to is a fine-adjustment mechanism. Its movements are all small. It would be instructive to know if there are any cases in which, with prolonged unconsciousness, there are recurrences of the characteristic muscular phenomena. Some of the protracted cases may signify the passing from one fit to another. In any case, as all authorities are agreed, the affection is in some sense epileptoid.

LITERATURE.—1. QUAIN'S *Dictionary of Medicine.*—2. HACK TUKE'S *Dictionary of Psychological Medicine.*—3. GOWERS' *Diseases of the Nervous System.*

Cataract.

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TECHNICALLY the name cataract is applied to any intransparency of the crystalline lens. Popularly only those lenticular intransparencies which by their distribution and their density cause more or less considerable impairment of visual acuity are looked upon as cataracts. It is also common to talk of cataract developing when the intransparencies in the lens are of the nature of progressive degenerative changes.

From a diagnostic point of view it is often important to distinguish between the "stationary" varieties of cataract in the technical sense and the "progressive" forms. The former are also called "partial" cataracts, whilst the latter eventually become "total," or practically total, cataracts.

FORMATION OF CATARACT.—The loss of transparency which causes a progressive cataract is invariably the result of some degenerative change. The extent, moreover, to which these degenerative changes go leads to different appearances. There thus come to be distinguished a number of different varieties of total cataract. Only comparatively few of these varieties have any practical importance. The importance which may attach to them practically has reference to the selection of the most suitable operation for their removal, and consequently also to some extent to the prognosis of the result of operative interference.

A number of terms are in use to designate the stage at which a progressive cataract has arrived. Thus in any particular case the cataract may be "incipient," "advanced," "ripe," or "over-ripe." The meaning of the first two terms is self-evident. A cataract is ripe when the whole of the lens has lost its normal transparency. As a rule the sclerosed central portion of the lens, which is met with after the age of about thirty, and which forms as age advances a greater and greater proportion of the whole lens, the so-called "nucleus" of the lens, does not at first participate in the loss of transparency. It is therefore the totality of the surrounding and more superficial or "cortical" portion of the lens which is the site of a degeneration causing the transparency to be lost.

When the whole "cortex" has become intransparent, then the cataract is said to be ripe. The term "ripe" has reference to the circumstance that the lens proper is then readily removed out of its capsule like a ripe nut from its shell.

The further progression of the degenerative changes which have caused the total intransparency lead to over-ripe cataract. Thus the cortex may become liquified or cretaceous. In addition, the lens capsule may in part (over an area corresponding to that left uncovered by a semi-dilated pupil) participate in the loss of transparency or the suspensory ligament of the lens undergo degenerative changes. These latter changes are of importance from the point of view of treatment.

When once the lens cortex has lost its transparency, it is extremely rare that the transparency is regained. Practically speaking, there is therefore no cure for cataract in the sense that the eye can recover its normal condition. Absorption of the altered lens substance may, however, take place spontaneously. In many cases this can be induced by surgical interference. When the intransparent lens is thus got rid of by absorption, the obstacle to the formation of retinal images is removed and sight is restored. The optical condition of the eye is, however, different from what it was with the transparent lines *in situ*. Generally the new condition is one which admits of less sharp retinal images. Sharp vision can then generally only be got by means of a glass. The object of most cataract operations is to get rid of the intransparent lens. In all cases of total cataract this is necessary.

Cataract may be either traumatic or idiopathic. However arising, it is essentially due to some defect in the nutrition of the lens fibres.

TRAUMATIC CATARACT is either the *direct* result of some interruption in the integrity of the lens capsule, or the *indirect* result of injury to other parts of the eye. In the first case the loss of transparency of the crystalline lens is due to its coming into direct contact with the aqueous humour. In the second case the injury to deeper parts of the eye leads to intraocular changes, with which are associated changes in the character or in the amount of the nutrient liquids which percolate the lens.

In order that the lens may remain transparent, it is necessary that the lens capsule should be normal. The smallest breach in continuity of this membrane leads to intransparency.

A cataract caused by a wound of the capsule makes its first appearance very soon, in a few hours at most, after the injury, and afterwards develops more or less rapidly according to circumstances. Very rarely, and only in cases in which the laceration of the capsule is very minute and has rapidly cicatrised, the intransparency of the lens which follows may remain partial, or even slowly disappear altogether. This rare result is only met with too in young children.

The laceration of the lens capsule is usually caused by a perforating wound of the eye. Occasionally a sharp blow on the eye may cause a rupture of the capsule, and the result is similar, viz. an immediate loss of transparency of the lens.

Such cases of capsular injury, either by perforation or rupture, cause then what may be called a direct traumatic cataract. In other injuries to the eye unassociated with injury to the capsule, the cataract which may follow is "indirect." An indirect traumatic cataract only makes its appearance weeks or months after the injury.

IDIOPATHIC CATARACT again may be either "primary" and uncomplicated, or "secondary" to some other disease which of itself has caused, or is likely to cause, serious interference with vision.

Of the primary cases, uncomplicated so far as any other defect of the eye is concerned, two groups may be recognised. The first group, fortunately by far the larger, includes all the cases in which there is no evidence of any general condition of malnutrition. In the second group there may be more or less reason on the other hand to connect the loss of transparency in the lens with some such condition as albuminuria, diabetes, etc.

Idiopathic cataract when primary and occurring in an otherwise healthy individual is most common after the age of sixty. On this account it is usually known as *senile cataract.* There is, however, no necessary connection between this variety of cataract and other evidences of senility. In many cases there seems to be a more or less evident hereditary tendency to cataract. On the whole it would appear, too, as if the younger the individual affected with cataract, the more evident was the hereditary predisposition.

Apart from heredity, which is by no means always traceable, there does not appear to be any general state associated at all intimately with the appearance of cataract. All that can be said is that on the whole there is a tendency increasing with age for the nutrition of the lens to become impaired, and that this tendency is greater in some families than in others. Probably the structure of the lens itself predisposes to defective nutrition. The lens is an epithelial structure, which, as time goes on, becomes more and more compressed and sclerosed in its central portion. This compression results from the continual growth of new lens fibres which go to form the outer layers, whilst the old effete tissue, not being removed, gradually accumulates. The oldest and most horny portions of the lens, instead of being rubbed off, as in the skin, are thus collected in its centre. This arrangement of itself seems to predispose to difficulties of nutrition. These difficulties are more insuperable in some individuals than in others, altogether independent of anything but mere local conditions. Whatever be the true and complete explanation, it is of practical importance to recognise that the term senile as applied to cataract has only a restricted sense. The appearance of cataract is therefore not necessarily, or even commonly, an indication of any general breakdown.

Most cases of senile cataract are cortical cataracts, *i.e.* the loss of transparency which takes place is limited to the cortex. In very old people the lens nucleus may become clouded, and then usually assumes a dark brown or almost black colour. This is sometimes, and especially when the colour is very dark, called black cataract (*cataracta nigra*). A better term is *nuclear senile cataract.* Although in the aged the nucleus of the lens comprises almost the whole of that structure, so that very little cortex remains, the true nuclear senile cataract not being such an essentially degenerative change, a nuclear cataract does not cause such a complete loss of vision as is caused by an ordinary ripe senile cataract.

DIAGNOSIS.—Mere inspection of the eye reveals a grayish opacity in the pupil. The pupil itself is freely movable, contracting as light falls into the eye, and dilating when the eye is shaded from the light. The history, too, is one of gradually increasing blindness until the details of objects have become indiscernible, and only the movement of large bright objects in front of the eye, or it may be only the difference between light and darkness, can be made out.

The earlier stages of cataract, as well as the differential diagnosis between visual defects due to intransparencies of the lens or to other causes, can only be made with the ophthalmoscope. Any intransparency in the dioptric media (the vitreous, lens, aqueous, and cornea) appears in the disc of *fundus reflex* (see "Eye, Examination of") as a dark area interrupting its continuity. When dark spots are seen in this way, the diagnosis of the site of the intransparency has next to be made. A preliminary examination of the cornea and aqueous made by *oblique illumination* enables us to see how far any intransparencies in these parts contribute towards the appearances seen with the ophthalmoscope. If there are no corneal nebulæ and the aqueous is clear, these parts of the eye can be excluded, and the question then is whether the dark areas interrupting the fundus reflex are due to intransparencies in the lens or vitreous humour. Opacities in the vitreous are often movable, *i.e.* move independently of the eye when once set in motion by eye movements. Lens opacities are, on the other hand, stationary. The differential diagnosis is usually easily made, although in some cases of opacities lying far forwards in the vitreous there may be some difficulty.

The pupil in old people often appears gray or greenish gray. It presents then a very different appearance from the dark black pupil seen in earlier life. This gray appearance is due to light reflected from the anterior surface of the lens. In young people the superficial layers of the lens have a refractive index very nearly equal to that of the aqueous. Consequently little or no light is reflected from the surface of separation between these two media. The greater hardness of the older lens, with its consequent greater refractive index, causes considerable reflection from the lens surface. This reflection is sometimes so great as to give the appearance on mere inspection of cataract. Inasmuch, too, as old people often complain of failing sight from other causes than cataract, it is important not to mistake the condition described for cataract. All that is necessary to make the differential diagnosis is the *dioptric* test with the ophthalmoscopic mirror. When no cataract exists the fundus reflex, so far as the lens goes, is seen undisturbed throughout.

Senile cataract is mostly met with in both eyes. Often the condition first begins in the one eye alone. The first appearances are only seen with the ophthalmoscope and with the pupil dilated. They are generally those of radiating peripheral intransparencies, the so-called *striæ*. The stage of peripheral striæ may last for many years without appreciably advancing. Generally, however, the striæ become continually more numerous, larger, and confluent. Only small chinks of clear cortex then remain. Often this gives rise to a multiplication of the images of objects seen—what is called *polyopia*. Sometimes, however, the vision remains amazingly good, though the changes are often accompanied by alterations in the optical condition of the eye. Weaker reading glasses are then often used, and the distant vision is improved, it may be by concave glasses, or if previously convex ones have been required, they may no longer be necessary.

The further confluence of the separate areas of intransparency leads to the whole of the cortex being involved, and then generally pretty soon to the cataract becoming ripe. The *period occupied in ripening*, however, varies very much. As a rule, from the time the striæ first become evident till the whole cortex is intransparent, there is an interval of time of from two to three years. But there are frequent exceptions to this, and mostly in the direction of a prolongation of the period of development. It is therefore not safe in any given case to venture upon too definite a prognosis as to the date when the cataract will be ripe.

There is often a very serious degree of blindness long before the cataract is ripe. The bearing of this fact upon the question of treatment is afterwards considered.

The stage at which operation for cataract is most satisfactory is the stage of ripeness. Interference is on the whole less satisfactory before this stage has been arrived at, and still less satisfactory when there is any marked degree of over-ripeness.

The most important point which has to be inquired into in the case of a ripe cataract is as to whether or not the cataract is uncomplicated. A ripe cataract, whilst it leads to the loss of any sense of form, so that surrounding objects are not seen, does not materially interfere with the sense of light. Consequently an individual with a ripe cortical cataract has a good appreciation of differences in degree of illumination. The light from an ordinary candle flame in a dark room can be seen, and the difference at once recognised when it is shaded by the observer holding his hand in front of it, so as to prevent its rays falling on the patient's eye, or the difference caused in illumination by reflecting into the patient's eye the light of a gas jet turned down to almost its lowest point and then removing the reflection, is at once appreciated. One circumstance, then, which would cause one to infer that the eye was otherwise healthy, notwithstanding the existence of a ripe cataract, would be the possession of a keen perception of light. On the other hand, a defective light sense must cause suspicion of deeper disease. When the light sense is very defective there can be no doubt of the existence of complication. And this is, of course, still more the case when the patient is unable at all to distinguish light from darkness. This condition is a complete contra-indication to operation, as no sight could then be restored by removing the cataract.

Another point which has to be tested is in the cataractous eye called the *projection.* Although unable to distinguish ordinary objects, an individual with ripe cataract, if the eye be otherwise healthy, should nevertheless have a fair idea of the direction from which a bright light comes. Thus the direction of a candle flame in a dark room, held close to him above or below or to either side of the face, can be made out. Although the intransparent lens is not capable of forming sharp images, it is able, even in cases of ripe cataract, to so far collect the rays, whilst scattering most, that a maximum illumination of the retina takes place in and around the place at which, under normal conditions, the image of the flame would be formed.

CONGENITAL CATARACT.—In addition to the forms of cataract already enumerated, there is a whole series of juvenile and congenital cataracts. These may be complete or partial, cortical or nuclear.

It is most important to recognise and to treat congenital cataracts as early as possible if they are either complete or so extensive and dense as to cause a serious impediment to vision. If they are not removed within the first few months after birth, the resulting vision is rarely good. Cataract developing in eyes which have once acquired good sight may remain for an indefinite period without interfering with the possibility of sight being restored on its removal. The immediate result of a successful operation in such cases is in fact the complete restoration of the sight, even if the cataract may have been there for forty years or more. There is no amblyopia from disuse. It is otherwise, however, where the early development through practice, which is necessary for the perfecting of the eye as a seeing instrument, has been rendered impossible. Later operation will admit of a certain degree of sight being acquired. But the acquiring of sight under these conditions is a slow, laborious process, requiring often much training.

It always stops short of perfection—often much short of it. Operations in the early months of life give the best chance. The necessity for early operation is the main practical point to keep in view in connection with such cataracts. Their diagnosis presents no difficulties, and the differences met with in different cases are only of scientific interest. They are almost invariably bilateral.

A complete cataract existing in one eye only of a child is almost certainly of traumatic origin. A careful examination of the cornea will usually in these cases reveal a scar, often a very faint one, owing to the rapid tissue changes of childhood. The scar indicates where the perforation of the eye took place. Curiously enough, parents and others who bring young children with traumatic cataract long after the accident which caused it, often deny that any accident has taken place. They usually admit it, however, when it is pointed out to them that the scar is visible. Sometimes, as for instance when the penetration has been made with the point of a fine needle or a fine pair of scissors, the parents may actually be ignorant at the time that so serious an accident has occurred.

One of the most frequent forms of juvenile cataract is what is called *lamellar cataract*. This is a partial stationary cataract. It is either congenital or developed very early in life. In lamellar cataract only some of the lens fibres, which are arranged in lamellæ, are intransparent; hence the name. The defect of vision which this variety produces varies in degree according to the position and number of the intransparent lamellæ. The pupil shows only a moderate degree of opacity, sometimes, indeed, so slight that it is difficult to diagnose the condition without the ophthalmoscope. The ophthalmoscopic appearance is characteristic. If the pupil be dilated it is found that a disc-shaped darkening of the fundus reflex occupies a central area of it. The shaded area varies in extent, appears darker at its margins, and is surrounded by a clear zone. Often the circular line corresponding to the margin of the opacity is broken by little protrusions, which on closer inspection are found to be caused by more peripheral intransparencies, which, as they embrace the edge of the main opacity, have received the name of "riders."

Lamellar cataract is rarely noticed before schooling has begun. Then it is found that the child's sight is less acute than normal. Objects are held closer to the eyes in order to be seen than under normal conditions. This attracts attention, and is usually supposed to indicate the existence of myopia. Often, indeed, and no doubt as the result of this too close application of the eyes, the condition actually does become complicated with myopia. Anything, in fact, which causes the persistent use of the eyes in early life for the seeing of objects which lie too near to them is apt to give rise to myopia.

The question which has to be determined in all cases of lamellar cataract is whether or not it is desirable to operate. As the operation consists of removing the lens in some manner or other, any power of accommodation must necessarily be lost after operation. The question to be considered then is whether the improvement in vision which the operation will cause is sufficient to counterbalance the risk of operation, the necessity for wearing cataract glasses, and the loss of accommodation. Obviously, there must be some limit for which the advantages and disadvantages are pretty equally balanced. This limit will vary, too, according to the individual views of different operators. Some even go the length of operating on one eye only, leaving the other untouched, so as to retain as far as possible both advantages, that of increased visual acuity in the operated eye, and retention of accommodation in the one which remains unoperated upon. This, of course, is done at the expense of binocular vision. It is rare that such a practice is to be recommended. Indeed, it should only be resorted to when one eye is considerably worse than the other, and at the same time the occupation of the individual makes it seem advantageous. In this respect each case must be judged on its merits.

A good practical rule to be guided by is to recommend operation when the visual acuity is less than $\frac{20}{60}$, and to advise the patient to have nothing done where the acuity is greater than this limit. Here, again, however, one must to some extent be guided by the wishes of the patient, when matters have been properly explained to him or his parents, as well as by the nature of the occupation which he follows or wishes to follow.

Lamellar cataract is almost invariably bilateral. It seems to stand in some relation to rickets, though in most cases the manifestations of rickets are not particularly pronounced. Often there is an absence of enamel on the teeth, especially the canines. Children with congenital cataract in any form are occasionally of more or less markedly weak intellectual development.

Treatment of Cataract.—Nothing is known to have any definite effect in checking the progress of cataract. The degenerative process seems as a rule to be so intimately associated with difficulties of nutrition depending upon the natural structure of the crystalline lens, and so rarely dependent upon general conditions of health, that it is unlikely that the progression of cataract could be influenced by general treatment. It is conceivable, no doubt, that the conditions of nutrition might be modified to advantage by local treatment. Hitherto, however, any treatment which has been adopted to *dissipate* cataract has been by secret remedies. The favourable results claimed for such remedies have never been substantiated. It is even doubtful if any ordinary use of the eyes in any way influences the process. This being the case it is hardly fair to impose upon one's patients, as soon as the beginning of cataract is diagnosed, all sorts of restrictions instead of allowing them to have the full use of their eyes as long as possible. Excessive, continuous reading, especially with a bad illumination, may perhaps be undesirable.

When cataract has advanced sufficiently to cause some marked interference with sight it is sometimes possible to get some temporary improvement *by the use of a weak mydriatic or miotic or by suitable glasses.* In many cases of advancing cataract it is found that patients complain of seeing very badly when facing the light, or generally, when the illumination is strong. In reading, for instance, they prefer sometimes an illumination which is otherwise unsuitable owing to being too feeble. The reason for this is that when the pupil is small the total area of the portions of the lens which remain clear is insufficient to give a bright enough retinal image. With a semidilated pupil, however, the number of rays going to form the image is greater. And if the more excentric pupillary portions of the lens are relatively free from intransparencies, the proportion of effective rays may increase at a greater ratio than the diminution in illumination, which causes the pupil to become less contracted. A still greater advantage may under these conditions be got by causing an artificial dilatation of the pupil. The greater number of rays thus admitted to form the retinal image is not counteracted by the diminished illumination necessary to admit of a natural dilatation of the pupil. This artificial dilatation of the pupil is best secured by the use of a weak solution of sulphate of atropine ($\frac{1}{4}$ gr. to ℥i.), which may be applied daily, or even once every second day. In advancing cataract this should always be tried. If it proves of use it may then be safely indefinitely continued. It is comparatively seldom, on the other hand, that a miotic by causing an artificial contraction of the pupil is of use in cataract.

Patients with cataract often suffer too from a disagreeable degree of dazzling. This is caused by the scattering of many of the rays which enter the pupil. These scattered rays, mixing with the regularly refracted ones, disturb the clearness of the retinal images, and produce this disagreeable sensation. It is for this reason that cataract patients rarely face the light, or when they do, walk with their heads bent forwards. They frequently too shield the eyes by holding their hands at the side of the face, or by shading the eyes in some other way. Sometimes spectacles in the form of stenopaic slits cause improvement in vision by diminishing the amount of this scattering.

When the cataract in one eye is further advanced than in the other, the same scattering of rays in the worse eye causes the vision with both eyes to be less perfect, and associated with more discomfort than when the better eye is used alone. It is then advisable to allow them to read with an obscure screen in front of the worse eye.

Another expedient which may be resorted to when the sight is insufficient to permit of reading with the proper correction at an ordinary distance is to give up any attempt at binocular reading and use the better eye alone. It is then provided with a glass of sufficient strength to focus it for a distance of 6 or 4 or even 2 inches instead of for 10 to 14 inches. The retinal images thereby obtained are correspondingly larger, and thus reading may be continued for a longer time than is otherwise possible. Reading with a hand magnifying-glass also serves the same purpose.

The various *operations* performed for cataract may be classed under three heads—(1) Reclination; (2) Discission; and (3) Extraction.

Reclination or *couching* does not seem to be practised in civilised countries. It is a very old operation, and the one which was at one time universally employed. The operation consisted in pushing a needle, entered through the sclera, forwards between the lens and ciliary body, and then, by raising the handle, pushing the cataractous lens backwards into the vitreous. This operation, though producing necessarily, in uncomplicated cases, an immediate restoration of sight, was abandoned owing to the frequency with which it was followed by inflammation or glaucoma. It must be remembered, however, that reclination was performed exclusively in the pre-antiseptic period. Indeed, not only were no antiseptic precautions taken, but some of the descriptions given of the operation make it clear that it was customary for some operators to dip the cataract needle into, or smear it with, substances which were more likely than not to be septic. The risk of reclination nowadays could certainly not be nearly so great as it formerly was. Nevertheless, as a surgical proceeding, it is inferior to discission or extraction.

Discission consists in opening the anterior capsule with a fine, sharp cataract needle, and then passing the needle deeper into the substance of the lens and stirring it up. The aqueous humour is then brought into direct contact with the substance of the lens, which is macerated, driven forward in flocculent masses into the anterior chamber, and absorbed. The absorption takes place through the channels, at the angle of the anterior chamber, by which the aqueous leaves the eye. If the discission has been free, flocculent masses of macerated lens matter readily escape out of the capsule and lie in the anterior chamber. There they gradually become lessened in size as absorption slowly proceeds.

In performing discission care must be taken to make a sufficiently large opening in the capsule. When the lens is stirred up the swelling which takes place as it becomes macerated leads to an extrusion of some of its substance through the capsule wound into the anterior chamber. If the wound in the capsule is not large enough, as is, for instance, the case if the capsule has been merely punctured before the lens has been stirred up, the intracapsular swelling which follows may cause more or less irritation of the eye due to increased intraocular tension. Apart from this, however, absorption does not under these circumstances take place so readily, or it may stop altogether. The operation has then to be repeated.

The cataract needle in performing a discission should be passed very obliquely through the corneo-scleral margin into the anterior chamber, thus forming a valvular opening. After having reached a little beyond the middle of the chamber, the capsule should be lightly cut by raising the handle of the instrument till the point of the needle comes in contact with it. The needle point is then drawn over the surface of the capsule, and made to tear a linear aperture in it. The instrument is then slightly withdrawn till its point lies again free in the anterior chamber, then again pushed in sufficiently to enable another section of the capsule to be made in the same way, but in a direction crossing the first at as great an angle as possible. In other words, the capsule should be opened by a pretty free crucial incision which does not pass too deeply. After this is done the needle is pushed into the lens and stirred about. Care must be taken not to perforate the posterior capsule, which may readily be done towards the periphery where the lens is thinner.

The operation of discission done with an aseptic needle causes very little reaction. Absorption, however, takes place very slowly, several months elapsing before the pupil is clear. Occasionally after three or four days glaucomatous symptoms may develop. In children this is often accompanied by sickness, and the eye becomes congested and painful. It is then necessary to relieve the tension by extracting some of the macerated lens from the eye. This is done through an opening made at the corneo-scleral margin with a bent keratome. The lens matter is removed through this incision along a curette (a small grooved instrument made for this purpose), with which the outer lip of the wound is depressed, and which is introduced a short way into the anterior chamber.

This operation of linear piecemeal extraction is very often made to follow a discission even when no symptoms of irritation have supervened. The removal of the cataract is thus hastened. When properly performed, and with due antiseptic precautions, it is a perfectly safe proceeding.

Operation by discission with or without a subsequent linear extraction is only suitable for cataract in young people. Different operators, however, recognise different limits of age, after which discission should not be done. It is, generally speaking, not advisable to operate in this way when the patient is more than twenty-five. Too much irritation is otherwise apt to be caused by the harder central portions of the lens. In any case the later a discission is done the more necessary is it to follow it up with a linear extraction. As a rule it is better to perform the extraction at once, and not subject the patient to two operations.

Discission followed by linear extraction is the most satisfactory operation for lamellar cataract in children. It is also the operation generally performed for traumatic cataract in young people.

Extraction of the opaque lens as a whole from the eye is the method of operating which still remains to be described. There are several different methods of extraction in use. These methods mainly differ in minor details. The general principle of cataract extraction is to remove from the eye as completely as possible the whole of the intransparent lens in a manner which is conducive to rapid primary union of the external wound made for the purpose. The wound must not only occupy a position which does not interfere on cicatrisation with vision, but it must be so placed that the edges lie in good apposition after the cataract is removed, and are also readily nourished. The site usually chosen for the wound is therefore the corneo-scleral margin. Some operators, however, carry the incision more or less decidedly into the cornea.

The operation of extraction is performed with a long narrow knife, the so-called *Græfe knife.* The incision into the eye is preferably made along the upper corneo-scleral margin, where it is afterwards mostly covered by the upper lid. In making the incision the knife is entered at the outer side at a point in the corneo-scleral margin on about a level with the horizontal tangent to a semi-dilated pupil. This is what is generally called the *puncture.* The knife is then carried rapidly across the anterior chamber, and a *counter puncture* made in the inner and upper corneo-scleral margin at a point just opposite to the puncture. The incision should lie throughout in the corneo-scleral margin, and should be made with as little sawing motion as possible. The wound should, in fact, be as clean-cut as possible, and occupy about one-third of the corneo-scleral margin. If the extraction be combined with iridectomy a portion of iris is next excised, the iris being drawn out through the incision either with iris forceps or hook, and then snipped across with a pair of sharp iris scissors. When the hook is used this can be done without causing any pain. After replacing the edges of the wound (coloboma) in the iris, should they be caught at all in the external opening the capsule is ruptured with the cystotome. The incision of the capsule is made in different ways by different operators. The main point, however, to attend to is that it should be sufficiently free. This is best effected by scratching the capsule several times, so as to make sure that the cystotome is not merely moved on the surface of the lens below the capsule without enlarging the opening first made in that membrane. The cataractous lens is then caused to become engaged with its upper edge in the external wound. This is done by pressure on the lower half of the cornea. When it has thus become engaged, further pressure, followed by gliding the scoop used for the purpose upwards, causes the lens to escape out of the eye. Particular care must next be taken to replace any iris that may be caught in the ends of the incision. This is best done with a soft caoutchouc or tortoise-shell *repositor*. In the process of delivery it usually happens that some of the soft cortical portion of the lens is rubbed off and remains in the eye. Subsequent gentle pressure on the cornea, which is most conveniently made through the lid, enables one to get rid of this. The pressure causes the external wound to gape slightly, and at the same time rubs the cortex through it. Before applying any dressing it is necessary to make sure that the lips of the wound are in good apposition, and that they do not entangle any capsule or cortex. The repositor should be run along between them, so as at the same time to flatten out any flap of conjunctiva which may have been cut in making the incision.

When extraction is performed without iridectomy, what is called the *simple* as opposed to the *combined* extraction, the external wound has to be made slightly larger. The pressure too must be rather greater, as the lens has to pass in front of the iris, which necessitates also making the wound gape to a greater extent. In this operation cortical remains are removed with the curette, and not by pressure, after the iris has been carefully replaced, should the pupil not of itself have recovered its circular shape.

The Question of Iridectomy or no Iridectomy.—Whether the simple or the combined operation is to be preferred is one upon which much has been said, and one upon which different surgeons hold different views. The advantages of the simple operation are, that no wound is made in the iris, that the cosmetic effect of a round pupil aimed at in this operation is better than that of a coloboma even when the coloboma is made upwards, and that peripheral attachments of the iris to the inner surface of the external wound are less common. The disadvantages are that cortical remains are not so easily removed after the delivery of the lens, and that prolapse of the iris into the wound is more apt to take place than when iridectomy is done. In addition, the circular mobile pupil aimed at is by no means always got. Further, the simple operation is not so easy, and on this account alone not to be recommended to inexperienced operators. There is no difference in the result so far as the acuity of the restored vision goes, which is got by the simple as compared with the combined extraction. The simple extraction is in other respects, however, the more ideally perfect one.

In making the somewhat larger section which is required in the simple extraction there is some difficulty often in preventing the iris from falling in front of the knife as the anterior chamber empties. The blade must therefore be kept pushed well forward, raising as it were the cornea from the chamber. A miotic should also be used immediately before making the section, so that as soon as the iris is returned the pupil may be contracted. The eye should be examined from half an hour to an hour after the operation is completed. If the pupil at this stage is found to be contracted and quite circular, there is practically no danger of iris prolapse taking place. If, on the other hand, it is not circular, but hitched up towards the wound, the round movable pupil aimed at will not be got, and there is in addition a strong probability that healing will be impeded and more or less unnecessary risk run by prolapse. Under these circumstances the best thing to do undoubtedly is to perform an iridectomy at once, and therefore convert the simple into the combined extraction. This is very much more satisfactory than any treatment of prolapse at a later stage in the healing process. In the experience of the writer this abandonment of the intended simple extraction is required in 10 per cent of cases in which it is begun. In this respect, however, individual differences must of course exist.

This is hardly the place to discuss fully the relative merits of the simple and combined operations. Some surgeons perform exclusively the former, and some exclusively the latter, whilst others again make a selection of cases which appear to them most suited for the one or other method of operation.

The writer after a very extensive experience of both operations would, however, offer the following advice to less experienced operators:—

1. Do not attempt the simple operation at all until sufficient experience of the combined operation has been obtained by the performance of two or three hundred extractions.

2. In the first simple extractions undertaken select cases of fully ripe cataract in, comparatively speaking, young individuals, say up to the age of sixty or sixty-five.

3. Do not hesitate to abandon the simple extraction aimed at whenever the conditions above described indicate a risk of more or less troublesome prolapse.

It occasionally happens that when the attempt is being made to remove the lens by pressure on the cornea after the capsule has been ruptured, vitreous humour presents at, or escapes by, the external wound. Pressure has then to be abandoned and the lens has to be taken out with the *vectis*. This instrument, which consists of a fine steel loop, is inserted behind the lens by being gently forced past its upper margin with a slight side to side movement. As soon as it has slipped far enough back it is pushed downwards so that the lens comes to lie upon it. The vectis is then slowly withdrawn with the lens, which is at first kept pressed up against the back of the cornea until it has so far escaped from the eye that it readily comes away altogether. After this proceeding, if the lips of the wound do not lie in good apposition, the iris scissors should be used to cut across any vitreous which protrudes.

This accident may occur from want of care in the use of the cystotome or by too great straining on the patient's part. The suspensory ligament is then ruptured. In some cases, especially where the cataract is over-ripe, it may be impossible to prevent. As it is a complication which cannot always be foreseen, a vectis should always be at hand when performing a cataract extraction.

A considerable loss of vitreous may take place without impairing the result of a cataract operation. Yet there can be no doubt that with the loss of vitreous, which is never replaced, the future of the eye is not so secure.

Another complication which often calls for some modification of the method of operating is the coexistence of capsular cataract. When the capsular thickening is not very extensive the extraction may be performed in the ordinary way. With a large, dense, capsular cataract, however, it is best to extract a portion of the capsule instead of merely incising it before the lenticular cataract is removed. This is done with capsule forceps, of which there are various patterns. Extraction of the portion of capsule must be done carefully by gently moving the forceps from side to side as it is being withdrawn after having obtained a good hold of the membrane.

AFTER-TREATMENT OF CATARACT EXTRACTION.—Though not absolutely necessary, it is a good precaution to take to keep the patient in bed for two or three days after operation. This is the best way of as far as possible securing rest to the eye. Where this treatment proves very irksome, as it is apt to do in very stout or in diabetic individuals, it is better not to insist upon it.

With regard to dressings different ideas obtain. Some surgeons do not tie up the eye at all. Others apply some sort of dressing and bandaging, which is retained for a varying time afterwards. Others again tie up both eyes. Cataract extraction is an operation in which, given a healthy eye, rigid antiseptic precautions as regards the instruments used, and a certain amount of skill, and particularly quickness in performing the operation, the tendency to healing is fortunately quite remarkable. On this account it is practically impossible to justify any claim of superiority for one method of after-treatment over any other which is not altogether irrational. The rule in such a case should be to err rather on the safe side. It is therefore probably on the whole best to tie up the eye which has been operated on for a few days. Three or four days is sufficient. The dressing, too, may be changed once a day and the eye examined at each dressing. The surrounding light should also not be excessive where it is possible to control this. The tying up of the other eye does not seem to secure any greater rest to the one operated upon, as the patient instinctively keeps it closed for the first few hours until healing is sufficiently advanced to prevent movement giving rise to any pain. As in many cases too such treatment is found to be very trying to the patient, it is better to avoid it.

The only exception that should be made as regards tying up the eye after extraction is when there is an evident risk of infection from local sources, as there is, for instance, particularly in patients suffering at the same time from blenorrhœa of the lacrymal sac. In these cases it is better to leave the eye altogether uncovered. Where this complication exists it is also a good plan to dust freely finely powdered iodoform on the wound immediately after the operation is completed. The purulent secretion should subsequently be carefully removed from the lids two or three times in the twenty-four hours by irrigation with an antiseptic lotion. In view of the risk of infection, too, the operation for a cataract which is complicated in this way should be done as quickly as possible. It is especially important that no prolonged effort should be made to remove cortex, as every moment and every movement of the eye may be supposed to increase the chances of micro-organisms settling in the wound.

As a general rule the anterior chamber is found to be reformed in a few hours after extraction, not infrequently indeed even within one hour. This means that after so short a time sufficient agglutination has taken place between the lips of the wound to admit of the retention of the aqueous humour. The cleaner the section and the more perfect the apposition the more rapid is this primary union. Hence the importance of a sharp knife and the absence of sawing movements in making the section, as also the final cleaning of the wound.

Very little reaction takes place as a rule. There is usually a little congestion merely of the vessels in the neighbourhood of the wound. This congestion increases slightly during the three or four days that the wound takes to consolidate. Any further reaction is due to various causes. The most important is infection of any kind during operation or from local sources before healing has advanced. Other causes are the irritation of cortical remains and antiseptics introduced by instruments. A superficial conjunctival irritation often follows from too prolonged tying up of the eye or from the too frequent use of antiseptic lotions.

Of the Results of Infection the worst and most dreaded is primary suppuration of the cornea. This generally leads to complete destruction of the eye. Occasionally it stops short of this either as the result of the local vital reaction or of energetic and timely antiseptic measures. An infection of this kind usually shows itself within the first forty-eight hours after operation, often indeed within the first twenty-four hours. The patient complains of pain, the lids are red and swollen, and on inspection of the eye a grayish infiltration is seen stretching towards the centre of the cornea from the wound. An attempt should be made to check this at once as soon as it shows itself. This may be done by applying the thermo-cautery along the wound or by the use of freshly prepared chlorine water or of pure liquid carbolic acid painted on the wound with a fine camel's hair brush. The wound may then be dusted with finely powdered iodoform and all dressings removed from the eye. If the patient be old and feeble it is well also to use stimulants freely.

Most cases of primary suppuration occur from infection which has its origin in the conjunctiva or tear sac. The cases most likely to go wrong in this way are those complicated with blenorrhœa of the tear sac, especially when this is associated with blepharitis. When there are no such evidences of local infection it is nearly always the case that micro-organisms have been introduced by the instruments. In pre-antiseptic times extraction failed from this cause in a very much larger proportion of cases than now. Yet even at the present time probably no surgeon is able to steer free of corneal infection altogether. No, doubt therefore, there are exceptional cases in which the vitality of the cornea is insufficient to withstand infections which under ordinary circumstances would be counteracted. With rigid antiseptic precautions, however, not more than one eye in two hundred should be lost in this way where the surroundings parts are healthy at the time of operation.

Severe attacks of *iritis and iridocyclitis* are also set up by infection. Such cases seem to originate, so far as is known, in the remains of the lens tissue. Lens cortex has been shown experimentally to be the most favourable tissue in the eye for the growth of organisms. The result of iritis when the inflammation remains limited to the more anterior portions of the eye is to leave a more or less dense screen, consisting of iris, capsule, and fibrinous exudation, behind which the vitreous may be more or less infiltrated. The contraction of the iritic exudation often leads to a greater or less obliteration of the coloboma, when an iridectomy has been performed, so that the pupil is drawn up towards the external wound. Occasionally an infected iritis after cataract extraction may lead to sympathetic mischief (*vide* "Sympathetic Ophthalmitis").

Lesser degrees of iritis are frequent. They do not cause any displacement of the pupil, though some synechiæ form. They are probably not of an infected nature, but caused mostly by the irritation of remaining cortex.

Glaucoma may also occur as a complication of the healing process (*vide* "Secondary Glaucoma").

A serious accident which sometimes occurs, and which cannot be foreseen, is copious bleeding leading to detachment of the retina, which is then often protruded through the external wound. This may occur any time within the first twelve hours after operation. Most commonly it takes place almost immediately afterwards. The eye has then to be enucleated.

Only when the pupil is perfectly free from cortical remains is the sight on proper optical correction as good twenty-four hours after operation as it eventually becomes. As a rule, owing to gradual absorption of the matter left in the eye, vision slowly improves, and only reaches its maximum after, it may be, a good many weeks.

In by far the largest proportion of cases the best results, so far as the vision is concerned, are only to be got by a second operation, which consists of *needling* the capsule. This operation, though very safe when properly done, should be avoided whenever the vision is sufficiently good otherwise for all practical purposes. Not only is there some immediate risk attaching to it, some chance of either inflammation or glaucoma, but clinical evidence certainly appears to support the view that an eye in which the vitreous has been disturbed cannot be regarded as quite so safe as it would otherwise be. In all cases, therefore, the vitreous should be disturbed as little as possible. This is best attained by making the desired opening in the capsule with a single cutting needle or fine knife. The instrument should be entered through the corneo-scleral margin at a point which lies outside of the cicatrix of the original extraction wound. Its point should then be passed through the capsule at the opposite side of the pupil and the cutting edge directed backwards. Finally, the handle of the instrument is raised, causing the blade to sweep in the opposite direction and cut an opening in the capsule. This should be done in such a way, too, as to prevent the needle passing deeply into the vitreous. In most cases where needling is done in this way it may be done in the course of a fortnight after the first operation. The capsule is then elastic and readily retracts so as to leave a gaping opening where it has been cut. The contra-indications to early needling are any abnormal degree of reaction following the extraction and the presence of much soft free cortex.

In cases where owing to iritis the pupil has been closed and drawn up with the iris fibres put on the stretch, the opening is best made with a pair of small scissors constructed for the purpose (iridotomy scissors). An opening, which should not be too small, is made with a keratome in the corneo-scleral margin and the scissors introduced, with the sharp-pointed blade pushed behind the iris and the blunt one in front. As soon as the blades have passed over to the opposite side of the anterior chamber a snip is made. The cut, being at right angles to the stretched iris fibres, admits generally of sufficient retraction of these to maintain an opening. In the worst cases a bit of the opaque screen has to be cut out. In all such cases, however, the prognosis is less favourable.

The large opening made in the eye for the extraction of the crystalline lens causes, as might be expected, some alteration in corneal curvature. This *operative astigmatism* has to be taken into account in determining the optical correction afterwards required for near and distant vision. It is some time—generally a good many weeks—before the amount of the astigmatism has become constant. The progress of cicatrisation leads to its getting gradually less and less for some time. The final astigmatism varies in amount in different cases. This depends partly upon how the section has been made and how healing has taken place, but also upon the previous state of the eye as regards astigmatism. The operative astigmatism more frequently counteracts than supplements a previously existing astigmatism, as it is the horizontal meridian of the cornea which is most curved after operation. When the factor of pre-existing astigmatism is taken into account it will be found that for the same operator the acquired astigmatism is much the same whenever healing has taken a normal course.

One question remains to be considered, viz. How far in the case of slowly developing cataract is it necessary or advisable to wait until the cataract is ripe before operating? This question would no doubt be answered differently by different surgeons. The writer can only here offer such advice as his own experience leads him to consider sound. It has to be remembered that many unripe cataracts, especially in old people, can be extracted just as readily and as completely as can ripe cataracts. In many cases, however, a good deal more cortex remains in the eye. The cortex is often not only less readily removed, but being transparent at the time of operation, cannot be recognised. As the time taken for the sufficient clearing of the pupil depends upon the absorption of remaining cortex, unripe extractions take on the average somewhat longer before useful vision is restored, while the risk of iritis of all kinds is proportionately increased. A much larger proportion, too, require the second operation of needling. Against these disadvantages have to be put those of having to remain for years without any useful degree of vision if one waits till a cataract is ripe.

An estimate of the relative importance of the *pros* and *cons* in this case can only be arrived at by personal experience. The following line of action is suggested here:—

1. Do not hesitate to operate on cataract that is unripe, provided the patient has otherwise healthy eyes and adnexa and the vision is so far reduced as to interfere seriously with comfort, *e.g.* makes the reading of ordinary type or the following of the ordinary occupation impossible.
2. Do not operate on unripe cataract of one eye as long as the other eye retains sufficiently useful vision.
3. Wait till the cataract is ripe in all cases in which the risks of extraction from any cause are evidently greater than usual.
4. Do not perform preliminary iridectomy or any other operation for the artificial ripening of the cataract.

The practical result of following these rules is that one operates on a much larger proportion of unripe than of ripe cataract, but that when one has to regret the failure of the operation from serious subsequent complications it is mostly in cases in which there was not much to be lost at any rate.

CATARACT GLASSES.—After extraction, the eye, if previously emmetropic, becomes necessarily hypermetropic. The glass which then corrects for a distance when placed about 15 mm. in front of it is one of 10·0 or 11·0 dioptres. That is, in the condition of *aphakia* (absence of the crystalline lens) the previously emmetropic eye has to be provided with a glass lens of +10·0 to +11·0 in order to see distant objects distinctly. The retinal images then got of external objects besides being sharp are larger than they were previously in the proportion of about 3 : 2. If the eye were previously hypermetropic a stronger lens would be required for correction, *e.g.* if the degree of hypermetropia were 5·0 D, the glass after extraction would require to be +16·0 to +17·0. If the eye were previously myopic the correcting glass on the other hand would have to be weaker, *e.g.* if the previous degree of myopia were 5·0 D the lens after extraction would have to be +6·0 to +7·0; if 10·0 D +3·0 to +4·0; if 15·0 D, +0·75 to +1·50; if 20·0 D, −1·50 to −1·0 approximately. In the last case, therefore, the operative hypermetropia would not altogether counteract the previous myopia.

With the absence of accommodation necessarily following the removal of the crystalline lens (*vide* "Accommodation"), a stronger glass than that used for a distance is always required for reading. As a general rule the reading glass should be about 4·0 D stronger; therefore in the case of previously emmetropic eyes +14·0 to +15·0. But this depends upon the distance at which the individual reads. With good visual acuity he may prefer to use a weaker reading glass. Considerable alteration in focus is also got by sliding the spectacles up and down the nose, as their effect is stronger the further they are removed from the eye. In this way what is tantamount to a certain range of accommodation is practically obtained.

Cataract lenses should always be of glass, not of crystal (pebble). Glass does not transmit the actinic rays to the same extent, and these rays are hurtful to the eye.

Cataract lenses diminish the field of vision very materially. On this account they should be worn as large as possible, compatible with not too great weight. As a general rule the best vision is only obtained with sphero-cylindrical lenses, that is lenses, one surface of which is spherical and the other cylindrical. This is on account of the astigmatism already referred to. Sphero-cylindrical lenses are necessarily heavier than ordinary biconvex spherical lenses.

When only one eye has been operated upon and the other is of little use, the spectacles may conveniently be made reversible, *i.e.* the one side provided with the distant and the other with the reading glass.

Cataract glasses should not be worn as long as there is much cortex left to be absorbed. In any case it is inadvisable to use them sooner than a fortnight after extraction. When used soon they have often to be changed after some months owing to the change which takes place in the amount of operative astigmatism.

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Catheters, Uses and Dangers of.

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Catheters are hollow tubular instruments for drawing off urine through the urethra. They are made of metal, of silk webbing, impregnated with resins, so that they become rigid when cold and are made flexible by heat, of celluloid, and of red rubber. Usually they are about eleven inches in length, but those for prostatic cases may be fifteen or more. The size of English catheters is arbitrary, ranging from one-half to thirteen or fourteen. French ones are graduated from one to forty, the number indicating the circumference in millimetres. Flexible catheters are straight; metal ones are bent up for the last three inches (or four in the case of prostatic instruments), so as to form a quarter of a circle four inches in diameter. The English, so-called gum-elastic, catheters are provided with a metal stilet, and if bent to any shape when warmed retain it more or less perfectly after cooling.

Catheters are cylindrical in shape, and made with a terminal opening at one end and a lateral one (the eye) at the other. The vesical end may be rounded, or it may have a flexible neck with a small terminal bulb; or it may be stiffened and bent upwards (coudé or bicoudé) to suit various conditions of the urethra. The end beyond the eye must be solid, so that there is no space to lodge decomposing urine. The eye must be sunken below the surface and have its edges well rounded, and the surface inside and out must be absolutely smooth, polished, and aseptic, or it is not possible to keep the instrument clean or sterile. The other end, if the catheter is of metal, may be bent downwards to prevent urine spirting over the bed-clothes; if it is flexible the end should be enlarged so that it may not slip accidentally into the urethra. The ordinary ivory caps with which they are provided are a mistake; they merely harbour dirt.

Catheters should be sterilised before being used, and should be kept either in aseptic catheter cases or wrapped up in a carbolised towel. Metal and rubber ones can be boiled or steamed, or soaked in carbolic solution of sufficient strength for some hours. The aseptic ones mentioned already can also be boiled without injury so long as they are kept straight, or they can be immersed in a saturated solution of boracic acid for twenty-four hours. Other kinds must be syringed through before and after using with solution of nitrate of silver or corrosive sublimate (1-500), or be exposed in an atmosphere of formalin vapour or of sulphurous acid gas for some hours. But if the interior is rough it is scarcely possible to make it aseptic. The lubricant must be soluble in water, such as boroglyceride or lanolin. If it contains any form of grease the catheter cannot be cleansed without soda. The addition of an antiseptic to the lubricant is useful to prevent it from decomposing, but it is of no good so far as the lining membrane of the urethra is concerned. A weak antiseptic is useless, a strong one too irritating.

Method of Use.—The best position for the patient is lying upon the back with the shoulders raised a little and the thighs slightly flexed and abducted. Clothing should be warm, but the umbilicus must be exposed. It is often easier to introduce a catheter if the patient is standing upright, but there is then always the risk of syncope. The glans penis and the prepuce should be washed first with soap and water. Then they should be sponged over with a solution of nitrate of silver or corrosive sublimate (1-2000), and the fossa navicularis thoroughly syringed out. The urethra harbours a number of micro-organisms which are always liable to be carried in by the catheter; but unless it is inflamed septic ones rarely penetrate beyond the fossa. If there is any urethritis the whole canal down to the membranous portion should be douched out with nitrate of silver (1-1000) by means of a syringe giving a recurrent stream before the catheter is introduced.

Flexible instruments are always to be preferred, and the softer they are the better. Metal instruments are for purposes of diagnosis or to overcome some difficulty which a flexible one cannot. A medium size (15 F.) should be tried first. If this fails a smaller one can be taken. When the canal is tortuous olivary catheters insinuate themselves the most easily. Coudé ones follow the roof, and are therefore most useful when there is a dilatation at the bulb or a median enlargement of the prostate. Force is quite inadmissible; the instrument must find its own way. If the catheter is a rigid one the change in the direction of the urethra as it winds under the pubes through the triangular ligament must be borne in mind, and care must be taken to depress the handle sufficiently. False passages, when they do not begin in front of a stricture, always start from this point, and generally run upwards and backwards between the prostate and the bowel. If the finger is introduced into the rectum the least deviation can be detected at once. In cases of enlargement of the prostate an English gum-elastic, which has been kept for some time over-curved upon a stilet, sometimes succeeds when others do not; or Hey's well-known plan may be tried of passing the catheter down to the obstruction, withdrawing the stilet, and at the same time gently pressing the instrument onward. In stricture cases a catheter with a very long and fine whip-like end beyond the eye will sometimes find its way. Small metal catheters require the greatest care, as the points are very sharp and the walls of the urethra very tender. When the object is to draw off a large amount of residual urine, and it is essential to prevent any organisms being carried into the bladder, an instrument like Melchior's should be used. This consists of two tubes, one gliding inside the other. The outer is of metal, and is sufficiently long to reach the membranous portion. Its vesical end is closed with a film of rubber held in place by a metal cap. As soon as this is in position, a smaller flexible one is pushed down through it and through the rubber film directly into the prostatic urethra, so that the catheter which enters the bladder is never brought into contact with the meatus or the mucous membrane of the anterior portion of the urethra.

Pain and Spasm.—The urethra is exceedingly sensitive, especially the deeper portion. The passage of an instrument through this is always accompanied by pain and sometimes by syncope. All instruments must be warmed. Under the mucous membrane is a stout layer of unstriped muscular fibre which often grips the catheter firmly, and prevents it for a time from being pushed on or drawn back; but the contraction always tires itself out in the course of a few minutes. A preliminary injection of a few drops of a 2 per cent solution of cocain will prevent both pain and spasm.

The passage of a catheter, especially for the first time, is liable to be followed by consequences, some of which, such as syncope and shock, are referable to the *reflex action of the nervous system*, while others are due to *the introduction of septic organisms* into the deeper part of the urethra and the bladder.

Syncope.—Syncope is not uncommon, and may be serious if in an old man a large amount of residual urine is suddenly allowed to stream out through a full-sized catheter while he is standing upright. Shock is rarely grave or general, but local shock, due to inhibition of some of the nerve centres in the lumbar portion of the spinal cord, is not at all uncommon. It may show itself by retention from inhibition of the centre which controls the act of micturition; or by hæmaturia from congestion of the kidneys due to temporary vasomotor paralysis, or even by suppression of urine. As the cause is a very transient one the effects nearly always subside without any active measures being necessary.

Septic Poisoning.—The effects which are due to the introduction of septic organisms may be either local or general. The former include the inflammatory affections of the urethra, bladder, epididymis, and prostate, which are so often met with after careless catheterism; the latter comprise the different forms of what is miscalled catheter or urinary fever. The bacillus coli is the most common septic organism, but the staphylococcus aureus, citreus, and albus, the streptococcus pyogenes, and the bacillus ureæ liquefaciens are all of frequent occurrence, alone and together. They may enter the urinary passages through the blood-stream or the lymphatics, invading the walls first; or they may come down from the kidneys in the urine, or ascend upwards from the meatus (especially in the case of women); but when febrile attacks occur after the introduction of a catheter, the organisms which cause them are nearly always carried in on the surface or in the eye of the instrument. It is for this reason that sterilisation is so important.

The *simplest form of urinary fever* is a rigor or shivering fit, due to absorption through some abrasion (or perhaps even through the unbroken surface of the urethra) of the toxins which have been formed by septic organisms growing in the urethra. It begins as a rule, not when the catheter is passed, but some hours later, after the first act of micturition. All of a sudden the patient is seized with intense prostration and chilliness, which is described as being felt inside. The skin becomes rough and livid. The face is pinched. The eyes look sunken, and are surrounded by dusky rings. The respiration is hurried and shallow, and the pulse small and frequent. From the first moment, even when the teeth are chattering and the patient shivering, the temperature begins to rise, and it continues through the period of dry, burning heat which follows until sometimes it reaches 106° F. In ten minutes or half an hour, according to the severity of the attack, profuse perspiration sets in, the face becomes flushed, the involuntary muscular fibre in the skin relaxes, and there is a sensation of profound relief although the patient is utterly exhausted.

Rigors do not occur even after such operations as internal urethrotomy if care is taken to render the instruments, the urine, and the urethra aseptic. They are more rare after operations upon old and dense cartilaginous strictures than after those upon recent ones, not because the germs are fewer, but because in the former absorption is not so easy. They occur, as a rule, not when the catheter is passed, but afterwards at the first micturition, because the passage of the urine down the urethra raises the pressure and forces into the circulation the toxins which have been formed in the interval. They seldom occur after external urethrotomy, because owing to the open wound there is no pressure. After lateral lithotomy they are very rare, even when the urine is septic, for the same reason. And the same thing explains what has been so often noted, that, while every attempt at dilating a stricture causes a rigor, free urethrotomy or rapid and complete stretching cures the patient at once without the least reaction.

There are, of course, accessory causes. Any slight chill after an operation upon the urinary organs, such as standing with bare feet upon cold oil-cloth, will precipitate a rigor; and patients who have been exposed to malaria are especially predisposed to such attacks. But these are not necessary, and there can be no doubt, from what has been learnt by experimenting upon animals, that the toxins are perfectly efficient by themselves, even though the amount which is driven into the circulation is infinitesimal.

The prognosis in this form of urinary toxæmia is good as a rule. In some, fortunately very rare cases, the collapse has been so severe that the patient has never rallied. This is recorded as having happened even after the passage of a catheter, but most of the cases have followed operations upon the kidney for septic pyelitis. The explanation is that the manipulation of the kidney during the operation forces into the open ends of some of the renal venules such a large amount of the toxins which have been lying stagnant in the pelvis that the nerve centres are simply overwhelmed. Even when there are two or three rigors in succession there is no great cause for alarm. It usually means the absorption of successive doses. But when the rigors continue after forty-eight hours, after the surface of the wound in the urethra has been glazed over, it becomes a question whether the organisms themselves have not entered into the blood-stream as well as the toxins, whether it is not a case of septic infection rather than of simple toxæmia.

True septic infection may, of course, occur after operations upon the urethra as after operations upon other parts of the body. There are no local symptoms, but the rigors recur again and again, generally irregularly, but sometimes with intervals as regular as in ague. The patient rapidly becomes weaker and thinner. Perhaps slight jaundice sets in. There may be an attack of diarrhœa with peculiarly offensive motions. The pulse becomes more and more frequent, and at length death ensues from exhaustion.

Another and equally common form of what is called urinary fever is more chronic and much more insidious. The most typical examples are met with in cases of enlargement of the prostate at the beginning of catheter life, especially in those in whom there is a large amount of residual urine. A catheter is passed and the bladder partially emptied. This is repeated one or twice a day for several days. There is no rigor or other symptom of note. The urine is clear, but the specific gravity is low, and the amount unduly large. On the fourth or fifth day the bladder becomes irritable, and the urine is found to be a little turbid, so that after standing a gray deposit of pus settles at the bottom of the vessel. The total amount may increase until it reaches seven or eight pints a day, or it may diminish. The reaction is generally faintly acid, but it rarely fails to become alkaline after the first few days. There are often a few hyaline casts, and sometimes there is a little albumin. There is no rigor though the patient may complain of feeling chilly. The temperature only rises one or two degrees, but the pulse increases in frequency and diminishes in strength; the tongue becomes dry and brown, especially down the centre; the appetite is lost; there is a tendency to nausea; the mind becomes a little unsettled, and then muttering delirium begins at night, and the patient sinks into what is called a typhoid state. Post-mortem the bladder may be found to be large, with thin walls, or small and rigid with thick ones, and perhaps sacculi projecting from them; but there is always evidence of recent acute cystitis. The ureters are dilated and full of offensive urine and pus. The pelvis of the kidneys is in the same condition. The mucous membrane is discoloured, with flakes of pus adhering here and there. The apices of the pyramids are eroded. The kidneys themselves are small and hard, and on section are seen to be studded with minute abscesses differing in shape and outline according to the anatomical arrangement at that particular spot. In a word, there is evidence everywhere of long-standing disease of the bladder and kidneys with, in addition, recent septic inflammation and suppuration. The symptoms are not due to shock caused by emptying the bladder. They are in part the result of chronic renal insufficiency, in part of septic poisoning. The fault is in the bladder not being emptied properly. A certain amount of urine, a liquid in which micro-organisms grow exceedingly well, is habitually left stagnant in its recesses. Day by day more and more organisms are introduced by the catheter, and at last septic cystitis and pyelonephritis result.

Treatment.—Nothing special need be said about syncope or shock when they follow the passage of a catheter or any other operation upon the urethra. Retention of urine can often be prevented by a subcutaneous injection of strychnia given at the time of the operation. If it follows in spite of this a catheter must be passed again, but not until it is clear that there will be no relief without. Suppression must be treated by subcutaneous injections of digitalin, cupping over the loins, hot baths, and purgatives.

So far as what is commonly called urinary fever is concerned, the main treatment is prevention. All instruments should be sterilised beforehand, and only touched with hands that have been rendered thoroughly aseptic. The patient's bowels should have been well opened, preferably by a purge containing calomel, and the urine should have been rendered as resistant as possible to living organisms by the administration of intestinal antiseptics for some days beforehand. Salol and naphthalin I believe to be the two most efficacious, but the former should not be given in doses larger than three or four grains at a time, for fear of forming intestinal concretions. The patient should remain quiet for some hours after the operation, even if it has been merely the passage of a catheter (unless he is accustomed to it), and great care must be taken to avoid anything that might cause a chill.

The bladder should be emptied at the time of the operation, and micturition postponed for as long as the patient conveniently can. There will then be less risk of absorption. A hot hip bath just before the first act of micturition lessens the chance of a rigor, not only by its action upon the skin, but by the way in which it causes all the unstriped muscular fibre around the urethra to relax, and so lessens the pressure as the urine is driven down. At the same time the patient may be given a cup of hot tea, with some brandy, opium, and quinine.

It is probably impossible to render the mucous membrane of the urethra really aseptic, certainly if it is inflamed; but there is some advantage, I believe, in washing it out thoroughly before such an operation as internal urethrotomy, either with a saturated solution of boracic acid or with nitrate of silver, one in a thousand. After a stricture has been divided in this manner I always inject on to the face of the wound twenty drops to half a drachm of a ½ per cent solution of nitrate of silver or of protargol, and leave it there. The same thing should be done at the neck of the bladder in cases in which a large amount of residual urine is evacuated. It certainly checks, if it does not absolutely prevent, the growth of septic organisms at the critical place and time.

If a second rigor occurs after internal urethrotomy or any other operation upon the urethra, the injection should be repeated, and a catheter tied in so that the bladder may be kept permanently empty for forty-eight hours. This renders further absorption almost impossible. It is not wise to leave the catheter longer than forty-eight hours for fear of causing urethritis.

If the bladder becomes irritable, or if any deposit of pus makes its appearance after the evacuation of residual urine, the same thing should be done; and in addition, if the state of the bladder will allow it, the bladder itself should be washed out twice a day with corrosive sublimate or nitrate of silver. If this does not succeed very soon, or if it is evident from the shape of the prostate, or the condition of the bladder, that it is not possible to keep the cavity empty by tying a catheter in, there should be no hesitation in adopting perineal drainage or even puncture through the prostate. If septic cystitis is allowed to continue pyelitis and pyelonephritis will follow. The most successful way of stopping inflammation of the bladder is to follow the plan adopted by nature so far as it can, and empty the bladder and keep it empty. The danger lies in leaving a collection of urine, which has become converted by septic organisms into a virulent poison, stagnant in the post-prostatic pouch. The wisest plan is to adopt adequate measures while there is yet time.

Very little can be done in these cases towards restoring the aseptic character of the urine by means of drugs. Unhappily, intestinal antiseptics have very little influence upon the urine. They may make it less favourable as a nutrient medium for the growth of organisms, but the amount that reaches the urine through the kidneys is not sufficient to kill organisms which have already established themselves. And it must not be forgotten that the majority of patients who are attacked by this form of urinary fever are already enfeebled by long-standing renal inadequacy, and that anything which tends to upset their digestion, or prevent their taking sufficient nourishment, is more likely to do them harm than good. Quinine, boric acid, salol, and naphthalin are the drugs which I have found most useful, but the patient must not be forgotten while the complaint from which he is suffering is being treated.

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Cauda Equina. *See* Spinal Cord.

Cautery.—There are three varieties of cautery: (1) The actual cautery, the application points being usually hatchet-shaped, globular, or flat; (2) thermo- or Paquelin cautery, the terminals being straight, curved, knife-like, pointed, or flat; and (3) galvano-cautery.

Heat at which employed.—This may be black, red, or white heat, and varies according to the object of its use.

Black Heat.—The cautery is used at black heat as a counter-irritant. This is useful in many chronic affections of *bones, joints,* and *serous membranes*, such as spinal meninges, pleuræ, bursæ, tendon sheaths, in chronic conditions of *spinal cord, nerves,* especially intercostal neuralgia and sciatica. It is also of service in hysterical conditions, with localised painful areas, and in cases of malingering.

Red Heat.—It may be used at a dull or a bright red heat. (1) *At a dull red heat* the cautery is used as a severer form of counter-irritant in the conditions mentioned previously, and also as a hæmostatic in cases of hæmorrhage where a ligature cannot be applied, *e.g.* bleeding from bone or tooth sockets, or oozing from a large surface, or in hæmophilic subjects. Occasionally it is required when tracheotomy has to be performed through a very vascular growth.

(2) *At a bright red heat* it is not applicable as a hæmostatic, the eschar being too weak. It is useful, however, as a severe counter-irritant, and also for touching fistulous openings, *e.g.* urethra, rectum, umbilicus, salivary ducts, with a view to encouraging them to close. Phagedænic sores and the surface of poisoned wounds are often much benefited by its use. It is also employed for the removal of urethral caruncles, prolapse of the rectum, growing capillary nævi, cirsoid aneurysm, and also ignipuncture of hypertrophied tonsils. The employment of the cautery for the removal of tumours is now abandoned.

White Heat.—This is applicable in all the previously mentioned conditions, except where the hæmostatic action is desired. It is the most useful heat to employ where tissue has to be punctured to some depth, as in ignipuncture of tuberculous glands, as it retains the heat for a longer time. Negreth has recommended its use in hypertrophy of the prostate, the gland being punctured from the rectum.

Technique.—1. *Actual Cautery.*—When *black heat* is to be used the button cautery, or Corrigan's button, is the form employed, a latch key making a good substitute. When the cautery has been heated in the flame of a spirit-lamp until the metal shaft close to the wooden handle becomes so hot that it only allows of momentary painless touching with the finger, it should be rapidly tapped over the painful areas. For *red or white heat* the cautery is placed in a fire, and on removal is applied whenever its temperature has reached the desired limit. The hatchet-shaped instrument is used for linear cauterisation, the parallel lines being drawn at a distance of about one inch apart. If the flat form be used it is drawn over the surface at the rate of about one inch a second. For hæmorrhage the bleeding surface is steadily seared by rubbing the cautery over it, otherwise the instrument may adhere, and in its removal separate the eschar, and the hæmorrhage may be again started. If the bleeding point is in bone or the socket of the tooth, a knitting needle at red heat bored against the point acts well.

2. *Sherrow Cautery or Paquelin's.*—Before use the instrument should always be carefully tested, special attention being paid to the following points:—The benzoline reservoir should be only half filled, and the benzoline should be fresh, as it rapidly loses the active and more volatile portions. The blade should be heated red-hot in a spirit lamp, and then the vapour pumped in. Should it not work, most probably the benzoline is at fault. and the heat of the hand over the reservoir may be necessary to distil over some active vapour.

It is used in the same way as the actual cautery, but has one great advantage, that is, when it becomes adherent to the tissues the temperature can be raised and the cautery removed without detaching the eschar. After use the blade should be allowed to cool and then be dried. In ignipuncture for a painful spine, the skewer-shaped cautery is pushed through the skin over the lateral soft tissues, and inserted about ¼ to ⅓ of an inch. The punctures are made about one inch apart.

The Dechery automatic cautery is another variety of the thermo-cautery. Here ether is used instead of benzoline, half an ounce being sufficient for forty minutes' incandescence. This is a very convenient and portable form of instrument.

Practical Points in the Application. — (*a*) When the cautery is employed at red or white heat a *general anæsthetic* should be administered, and if ether be used great care must be adopted lest the inflammable vapour become ignited by the cautery.

(*b*) If the *constitution of the patient* is enfeebled by anæmia, Bright's disease, diabetes, melancholia, or other cause, the use of the cautery at red or white heat should be avoided, the reparative power of the tissues being much diminished. For the same reason it ought not to be used, if from disease or from injury the trophic power of a part is impaired.

(*c*) *Area of Application.*—All hairy parts should be shaved, and the surface rendered aseptic. All open surfaces, as sores, fistulæ, should be dried, and any blood-clots present removed. If the cautery is to be used for localised tender spots they should be marked before commencing the application. Soft parts with thick skin are the most suitable surfaces for application.

When superficial all bones, nerves, tendons should be avoided; also the trachea and urethra. In brunettes, the neck, arms, and face are better avoided as permanent pigmentation may result.

(*d*) The Local Results.—The effect at first is very painful, especially when red and white heat have been applied. The application of the cautery produces burns of various degrees at the point of application, and "radiation burns" in the immediate neighbourhood. The eschar following burns of the third and fourth degree may take several days to separate. When the cautery has been employed as a hæmostatic, the separation of such eschars are apt to be followed by hæmorrhage. After ignipuncture the healing may be very slow.

After-treatment.—If there be much pain anodynes internally may be given, except in hysterical patients and malingerers.

In burns of the first and second degree a weak antiseptic ointment spread on lint is all that is required (see "Burns").

In severer forms of burn cold boracic compresses relieve the pain. If delay in the healing is desired, ung. sabinæ two parts to vaseline one or two parts should be rubbed in once a day, and if the pain so induced be very great 2 per cent to 10 per cent cocaine may be added. The eschar being aseptic does not interfere with the healing of a wound by primary union. The disagreeable odour of burning flesh may be partially concealed by the fumes obtained from placing the red-hot cautery in some ground coffee.

Cellulitis. *See* Suppuration.

Cervix Uteri. *See* Uterus.

Cephalhæmatoma. *See* Head, Injuries of.

Cerebellum, Affections of.

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Experimental Physiology.—The striking feature of all experimental lesions of the cerebellum or of its peduncles is *disturbance* of *equilibration.* The results of removal of one lateral lobe, including the flocculus, are practically the same as removal of the corresponding half of the middle lobe as well, except that in the latter case the phenomena are more pronounced and persist for a longer time. The increase in the amount of disturbance is quite out of proportion to the additional amount of cerebellar tissue removed, so that the middle lobe appears to be of more functional importance than the lateral lobes. After unilateral ablation the disturbance of equilibration is manifested in some animals by a tendency to rotation about their long axis. The direction of this rotation is best described in terms relating to a right-handed male screw, the animal being considered as a screw, and its head as corresponding to that of the screw. After removal of the right half of the cerebellum the rotation is as if such a screw were entering an object, while in left-sided ablation the rotation is as if the screw were coming out of an object. Whether this phenomenon be present or not there is marked unsteadiness, and the inco-ordination may be so great that station and locomotion are made impossible. When progression is attempted the loss of balance may be evidenced by a pitching or falling away from the side of the lesion ; but from this must be carefully distinguished a tendency to fall to the side of the lesion in consequence of inco-ordination and motor paresis in the limbs on this side. Ferrier and Turner do not admit that any true paresis results, and like them I have been unable to find an efferent path direct from the cerebellum to the spinal cord, but their results accord with my own in tracing an efferent path through Deiters' nucleus, the tract occupying the antero-lateral region of the spinal cord on the same side in the position assigned by Marchi to the tract supposed by him and certain other observers to pass directly from the cerbellum and to degenerate after lesions limited to the cerebellum. I have shown that the *motor disturbances* met with in the limbs on the same side as a lesion of the cerebellum may possibly find explanation in the disturbance of the opposite cerebral cortex which results from a unilateral ablation of the cerebellum. That it is on the other hand quite possible that efferent impulses normally reach the muscles of the limbs from the cerebellum by way of the path that has been traced from this organ to the spinal cord through Deiters' nucleus, and that to cutting off of such impulses is to be attributed the paresis which results on cerebellar ablation, is made evident by the results of division of the inferior peduncle together with the path from the cerebellum to Deiters' nucleus.

The *attitude* after removal of one half of the cerebellum is very characteristic, and consists in curving of the neck and spine with the concavity to the side of the lesion, in addition to which the head is twisted on the neck in such a way that the side of the face corresponding to the side of the lesion is directed upwards. That the curving of the spine is the result of spasm of the muscles on the side of the concavity seems clear, in that attempts to unbend the spine meet with distinct resistance, and on desisting the spine immediately becomes arched as before. *Spasm* is also evident in the muscles of the limbs on the side of the lesion, notably the fore limb, which is commonly extended in tonic spasm. The immediate effect noted in regard to *the tendon jerks* after the influence of the anæsthetic has passed off is an increase of the *knee-jerk* on the side of the lesion and diminution of that on the opposite side; but in the course of forty-eight hours the opposite knee-jerk also becomes exaggerated to a degree that may make it impossible to say which is the more increased of the two. In the course of some weeks the knee-jerks gradually become less active, and while that on the side of the lesion remains more active than normal, in two or three months the opposite knee-jerk has returned to about normal.

It is admittedly very difficult to test the condition of *cutaneous sensibility* in animals; nevertheless, in my experiments I was able to determine what appeared to be distinct blunting of sensibility, most marked in the limbs on the side of the lesion, and present in lesser degree in the posterior limb of the opposite side.

The *ocular displacement* which results from unilateral ablation is, according to Luciani, one in which both eyes are turned to the opposite side. I have not been able to satisfy myself that there is much displacement of the eye on the same side as the lesion, but the opposite eye is always turned outwards and downwards to a variable degree. There is also nystagmus, which is most marked when the animal is induced to look towards the side of the lesion, and which consists in lateral jerking of the globes.

So striking and pronounced are the disturbances of equilibration and other defects consequent on experimental lesions of the cerebellum, that it is most surprising how rapidly compensation is effected, for in the course of two or three months such animals might pass as normal, even to the experienced eye, until certain minute examinations are made. Luciani has, however, shown that such compensation is not effected if the sensori-motor cortex of the Rolandic region of the cerebrum be also destroyed.

The cerebellum is prone to the same diseases as attack other parts of the central nervous system. Hæmorrhage, softening, abscess, tumour, developmental defects, atrophy, and degeneration are all met with, but the frequency with which they occur varies greatly, both as compared one with another and with similar affections of the cerebrum. Hæmorrhage and softening from vascular occlusion are infinitely more common in the cerebrum than in the cerebellum, whereas the latter organ supplies no small proportion of the total number of tumours and abscesses that occur within the cranial cavity. Indeed, bulk for bulk, the cerebellar tissue seems more liable to engender new growths than does that of the cerebrum.

HÆMORRHAGE into the cerebellum is a rare condition, is as a rule large, and commonly ploughs up the lateral lobe of one side, much of the middle lobe, and may even encroach to some extent into the opposite lateral lobe. The blood may, further, make its way beyond the limits of the organ either by way of the middle peduncle into the pons, to the surface, becoming subarachnoid, and spreading up under the tentorium, or it may burst into the fourth ventricle. Small hæmorrhages have usually been found in the region of the nucleus dentatus. It is exceptional to meet with a primary hæmorrhage of the peduncles, although the middle peduncle may become involved in the way already described, or blood may extend for some distance along its fibres as a result of a hæmorrhage in the pons. The branch which the superior cerebellar artery supplies to the corpus dentatum is that which usually gives way, though rupture of a branch of the posterior cerebral artery has been responsible for extravasation into the cerebellum.

A cerebellar hæmorrhage may cause rapid coma, terminating in death;

but on the other hand, premonitory symptoms have been known to commence as long as forty-eight hours before the definite evidence of onset. The most notable of these have been headache, giddiness, and turning of the head to one side. Headache is also a prominent symptom of the actual attack; the pain is felt suddenly at the back of the head and neck. Oft-repeated vomiting is another symptom that occurs early, and which is very rarely absent, while vertigo is also common. Rotation, which is an exceedingly rare symptom of cerebellar disease in man, has been met with more commonly as a symptom of hæmorrhage than of any other affection of the organ. The extravasation has been found in the middle or superior peduncle, or in one hemisphere, and the direction of rotation is said to have been from right to left when the hæmorrhage has been on the right side of the cerebellum, and from left to right when it has been on the left side. In the absence of actual rotation there may be a fixed position towards the side of the lesion with the head turned to that side. In patients that have been able to walk, a reeling gait or a tendency to swerve to one side instead of preserving a straight line has been noted, and with hæmorrhage in one lateral lobe there has been a subjective feeling of falling to the opposite side. The pupils are as a rule contracted, though they may dilate before death; the globes may deviate and may lose their parallelism, and there may be constant rapid nystagmus. Nothing constant has been found as regards the loss of motor power in these cases; in some there has been none, while in others hemiplegia has been present, but with no uniformity as regards the side affected, for sometimes it has been on the same side as the lesion, whereas in other cases it has been on the opposite side; a state of things that is possibly accounted for by the fact that the hæmorrhage is sometimes limited to the cerebellum, while in other instances it involves the pons as well. Priapism has been observed in cases of hæmorrhage into the middle lobe, but is probably due to secondary interference with the pons and medulla.

Softening is also rare, and in consequence of the free anastomosis of the vessels of the cerebellum, only a limited area is usually affected. The softening may result either from thrombosis or embolism, and is most frequently met with in the area supplied by the posterior cerebellar artery, in which the thrombus may originate, or there may be extension of a clot to this artery from the vertebral.

Necrotic softening of the cerebellum may occur without giving rise to any symptoms, while in cases in which the cerebellum is only one of the parts affected in consequence of occlusion of the vertebral, any symptoms that may result from the cerebellar condition are obscured by the more urgent phenomena that result from affection of the pons and medulla. But when a cerebellar artery alone has been occluded, inco-ordination of movement has sometimes been a symptom, as has hemiplegia on the same side as the softening, in one case accompanied by weakness of the facial muscles on the hemiplegic side.

Abscess.—Ordinary and pyæmic abscesses may of course occur in the cerebellum as in the cerebrum, but a detailed description of these conditions is unnecessary, as they have been dealt with in the article on inflammatory conditions of the brain.

Tumour.—Tumours are very common in the cerebellum, notably tubercular, gliomatous, and sarcomatous growths. Cysts are also met with, and when not parasitic or the result of old hæmorrhage, they are usually consequent on degeneration and breaking down of a new growth; simple cysts are rare.

Tumours may be situated in any part of the organ; they are commonly found deeply seated on one lateral lobe, or the middle lobe may be chiefly involved. A not uncommon position for growths originating in the region of the flocculus is on the external surface lying between the cerebellum and pons, while growths originating in the cerebellum may extend along its middle peduncle and infiltrate the pons and medulla secondarily. Tumours more or less limited to one or other of the peduncles of the cerebellum have been only very exceptionally met with.

Symptomatology.—As we are not here concerned with abscess of the cerebellum, and as the symptoms which result from hæmorrhage and softening have already been alluded to, the following account of the symptomatology of cerebellar disease, while not wholly inapplicable to other affections of the organ, is intended to relate mainly to tumours, while the manifestations which attend atrophy of the organ are reserved for subsequent and separate consideration. Sufficient has been said on these subjects in the article on intracranial tumours to make it unnecessary to refer to headache, vomiting, vertigo, optic neuritis, the effects on the pulse and respiration, and coma.

Experimental physiology has taught us two facts with regard to the cerebellum about which there can be no cavil; the one is that lesions of the organ result in disturbances of equilibration, and the other that the influence which the cerebellum exerts on the muscles of the body is mainly at any rate direct, and not crossed as in the case of the cerebrum, so that a lesion on one side of the cerebellum is responsible for certain defects in the limbs mainly on the same side. Disease of the organ in man amply bears out the experimental findings in regard to the first of these points, but in regard to the second, evidence of a most conflicting character is forthcoming; nevertheless it behoves us to keep prominently in mind during our clinical investigations that a direct relationship between the cerebellum and muscles does exist, so that we may give due weight to any clinical evidence which appears to accord with this fact.

It seems scarcely credible that an organ, lesions of which commonly cause profound disturbances of equilibration, may be the seat of the disease, with practically no indication that such exists; nevertheless this is so, and, moreover, experimental physiology prepares us for what would otherwise seem to be an anomaly. So rapid and so perfect is the process of compensation after acute lesions of the organ that we can understand how disease which produces its effects slowly may allow of substitution and compensation taking place *pari passu* with the effects of destruction, so that the symptoms expected of such lesions may never appear. The congenital and other forms of atrophy of the cerebellum may be thought to militate against this view, in that disturbances of equilibration are so common and so persisting in such cases; but in reality that this is to be accounted for by the concomitant defects present in the cerebrum, in these cases is proved by the experimental fact established by Luciani that compensation is not effected after cerebellar lesions if the sensori-motor cortex of the Rolandic region of the brain is also destroyed.

Disturbances of Equilibration.—Both the most common and the most characteristic symptom of tumours and other affections of the cerebellum is some disturbance of equilibration. The patient is usually unsteady on standing, and further evidence of loss of power of maintaining his equilibrium is manifested by the mode of his progression, as the unsteadiness is then at its maximum, and the patient reels and staggers like a drunken man. Such persons walk with their lower extremities far apart, and the movements of the limbs are performed in an irregular and hasty manner, as if attempting to prevent the tottering trunk from falling.

There is usually no constancy with regard to the direction in which patients with cerebellar tumour tend to fall; sometimes it is to the side of the lesion, and sometimes to the opposite side, or it may be in a backward or forward direction. Moreover, there is often no uniformity even in the same subject, there being a tendency to fall in different directions at different times.

Rotation as a symptom of cerebellar tumour, though not unknown, is exceedingly rare. Forced movements which compel the patient to lie on one side, so that if he is placed on the other he immediately returns to his former position, are not quite so rare; but there has been no constancy as regards the side on which the patient has been compelled to lie in consequence of the forced movement preventing him from lying on the opposite side. In fact the same patient has at one time in the course of his illness been compelled to lie on one side, and at some subsequent period it has only been possible for him to lie on the opposite side. These forced movements and rotation are indicative of lesions of the superior and middle peduncles of the cerebellum more commonly than of the organ itself. Although both rotation and forced movements are so rare, it is not so uncommon to meet with a fixed attitude which may be of some diagnostic significance; the head is inclined to one side, so that the ear and shoulder are approximated, while there is some rotation of the head, causing the chin to point to the side opposite to that to which the head inclines. There may also be lowering of the shoulder with slight curving of the spine, with the concavity of the arch on the side to which the head is tilted. Certain cases in man corroborate the experimental evidence on this point in that the tumour of the cerebellum has been found on the side to which the head inclines, but the sign is robbed of much of its value by the fact that the neoplasm is sometimes found on the opposite side of the cerebellum, and in other cases in the cerebrum it may be in some part so far forward as to preclude the possibility that the attitude is the outcome of pressure on the superior peduncle of the cerebellum.

Tremor is not a common symptom in cases of cerebellar tumour; nevertheless it occurs in the head and limbs, especially the superior extremities in some cases. It may resemble that seen in disseminate sclerosis, and when unilateral, may affect the arm on the side of the lesion; but too much reliance must not be placed on this sign in localisation, for in some of the recorded cases unilateral tremor has been on the side opposite to the lesion.

Motor Paralysis.—The majority of cases of tumour of the cerebellum are unattended by any detectable paresis or paralysis of the limbs, but in some cases such defects are met with. In some of the patients the distribution of the paralysis is hemiplegic, in others paraplegic, or the latter variety may be associated with weakness of one arm; so, too, the arm on one side may alone present evidence of loss of power, or when it is affected in conjunction with the leg on the same side, the loss of power in the upper extremity may be considerably in excess of that of the lower limb. When a hemiplegia or brachial monoplegia exists, it is sometimes on the side of the lesion of the cerebellum, while in other cases it is on the opposite side, though the latter seems less likely to be the case when the arm alone is weak. The explanation usually offered for hemiplegia of the opposite side is that it is due to pressure on the pyramidal tract above the decussation of the pyramids, while in explanation of the weakness that is sometimes met with in the limbs on the same side as the cerebellar lesion several explanations have been suggested. Those who deny that motor paresis can result from a defect of the cerebellum alone have supposed that such remote and improbable possibilities as counter-pressure of the opposite crus cerebri against the edge of the tentorium and petrous portion of the temporal bone, or direct pressure on the pyramidal tract below the decussation, are responsible for the defect, neither of which explanations can account for the weakness of the limbs on the same side as a necrotic softening of the organ consequent on vascular occlusion, cases of which kind are on record. My own experience, which is based on the results of experiments, leads me to believe that in such cases the weakness is directly due to the defect of the cerebellum. The paresis is so quickly compensated after experimental lesions of the organ, that it is not surprising that this symptom is not more commonly observed as a result of cerebellar tumours, for they may produce their effects sufficiently slowly as to allow of complete suppression, owing to substitution and compensation, even of symptoms that are usually pronounced. Dr. Hughlings Jackson has long contended that true motor weakness results from destructive lesions of the middle lobe of the cerebellum in man, and that the trunk muscles are first and most affected. We have published jointly a case in point, in which weakness of the spinal muscles was observed in a patient in whom there was cystic destruction of the middle lobe and adjacent parts of the lateral lobes of the cerebellum. As a rule, however, no weakness of the spinal muscles can be determined in cases of cerebellar tumour.

Convulsions.—When convulsions occur in a case of cerebellar tumour they are usually general, and are probably to be attributed to some indirect influence on the cerebral hemispheres. Unilateral convulsions have, however, been noted by Drummond in a case of abscess, in which the face and limbs were convulsed on the side of the cerebellar lesion. Peculiar attacks of tetanic-like spasms in cases of cerebellar tumour, causing retraction of the head, flexion of the forearms, extension of the lower extremities, and pointing of the toes, have been recorded by different observers, including Dr. Hughlings Jackson, who regards these attacks as of cerebellar origin. Others have, however, been of opinion that these seizures are due to pressure on the subjacent pons and medulla, the more so as the tumour has been in the middle lobe of the cerebellum in the cases described.

Tendon Jerks.—The knee-jerks afford us practically no information which can be regarded as of localising value in tumours of the cerebellum. They may be normal, exaggerated, or absent in different cases, or great variability of the knee-jerks may be observed in the same case; they may be increased, then diminished, it may be almost to extinction, and subsequently become comparatively brisk again. It was at one time supposed that absence of the knee-jerks in tumours of the cerebellum was of diagnostic significance, but it is now recognised that the sign has no such value, for the knee-jerks behave in a similar way in certain cases of tumour of the cerebrum. Moreover, it is worthy of note that an observer of Oppenheim's experience has never met with absent knee-jerks in an uncomplicated case of cerebellar disease. Inequality of the knee-jerks might have been expected to supply some information as to the side of the tumour, for with inequality after experimental lesions the increased knee-jerk is on the side of the ablation of the cerebellar tissue; but while the same has been found in some cases of unilateral cerebellar disease, in others a diminished or almost absent knee-jerk has been met with on the side of the lesion.

Anæsthesia.—It is quite exceptional to meet with any defect of cutaneous sensibility as a symptom of cerebellar tumour in man, and when present it has usually been of a slight character, limited to the upper extremity, and affecting the hand chiefly. Most of the cases in which anæsthesia has been met with accord with my experimental results, in that the defect of sensibility has been detected on the side of the lesion, but it has also been observed on the opposite side, and the total number of cases in which the defect has been noted at all are too few to allow of any importance being attached to the information to be derived from them, more especially in view of the possibilities of indirect effects from pressure. Muscular sense is said to have been disturbed in a few cases, but this again must be an exceedingly rare phenomenon in cerebellar disease.

Affection of Cranial Nerves.—The evidence afforded by affection of certain of the cranial nerves is of great value, more especially in deciding on which side of the cerebellum a tumour is situated, for in the majority of cases in which there is evidence of unilateral involvement of any of these nerves, the tumour is situated on the side on which they are affected.

Olfactory Nerves.—Anosmia is a symptom in some cases of cerebellar tumour, and it may be unilateral, in which case the loss of smell usually corresponds to the side on which the tumour is situated. The possible mode of origin of this defect has been discussed in the article on tumours of the brain.

Optic Nerves.—In no region of the brain is a tumour so commonly attended with impairment of vision except when the optic tracts or chiasma are involved; but this is probably only due to the frequency and severity of optic neuritis, with post-neuritic atrophy, in cases of cerebellar tumour.

Ocular Nerves.—As squints may depend on more than one cause in cerebellar affections, it is important to recognise what ocular displacements may be the outcome of pressure on the nerves supplying the eye muscles. That the eyes may be turned towards the opposite side after an acute lesion of one-half of the cerebellum in man, I have satisfied myself by personal observation of a case of abscess of the cerebellum, and of another patient in whom some cerebellar tissue was removed from one lateral lobe of the organ in an attempt to enucleate a tumour. But it appears equally clear that in more chronic cases, such as tumours of the cerebellum, ocular displacements are the result of pressure on one or other of the nerves supplying the eye muscles, or it has been suggested that pressure on the corpora quadrigemina may account for some of the squints met with. The external rectus of one or other or of both sides is most commonly paralysed, and when the defect is unilateral the muscle on the side of the tumour is that which usually suffers, though it is not always so, as the opposite external rectus may be the first to succumb. The internal rectus on the same side as the tumour has been affected alone in a few cases, and it has also been affected on the same side in conjunction with the external rectus of the opposite side, the joint effect being turning of both eyes towards the side of the lesion, with inability to turn them to the opposite side. In other instances the patient has been unable to turn the eyes to the side of the lesion, or both eyes have been turned away from that side in consequence of weakness of the external rectus on the side of the lesion, and of the internal rectus on the opposite side. In estimating the value of the lateral displacements of the globes as indications of the probable seat of the tumour, it must be remembered that inability to turn the eyes towards the side of lesion may be a consequence of secondary extension of the growth of the pons. Defects of upward and downward movements of the globes have been noted, as has partial ptosis, sometimes on the same side as the tumour, but in other cases on the opposite side. Practically complete ophthalmoplegia has been met with in a few cases, but exophthalmos is rare. Nothing constant has been determined with regard to the pupils, for though commonly unequal, the larger pupil is sometimes on the same side as the tumour, sometimes on the opposite side. What has been said as regards the frequency of blindness in patients with cerebellar tumour prepares us for the number of cases in which the pupils are dilated, and inactive or sluggish to light. Nystagmus is a common symptom of cerebellar tumours. The cases that allowed of my studying ocular displacements in acute lesions of the cerebellum in man also enabled me to determine that the nystagmus corresponds in every particular with that which results from experimental ablation of one lateral lobe of the cerebellum, and that it consists in coarse lateral nystagnoid jerks, most marked when an attempt is made to turn the eyes towards the side of the lesion. No such uniformity obtains, however in regard to the results of tumours of the cerebellum, for in addition to any rotatory or irregular oscillation that may be present, it frequently happens that the lateral nystagmus is most marked when the eyes are turned away from the seat of the lesion.

Fifth Nerves.—The fifth nerve is not uncommonly affected in cases of cerebellar tumour, the resulting motor or sensory disturbance being on the same side as the tumour, though the defects have been bilateral in exceptional cases. Anæsthesia of the cornea is sometimes present on the affected side, and neuro-paralytic ophthalmia may result. Moreover, it is probably through the agency of pressure on these nerves that loss of taste is induced in a few cases. The affection of taste has usually been unilateral, and has corresponded to the side on which the tumour has been found.

Facial Nerves.—Unilateral facial paralysis or paresis is common, and, as Dr. Beevor has pointed out, evidence of implication of the seventh and eighth nerves indicates that the growth is probably in the anterior part of the posterior fossa as opposed to its being in the so-called cerebellar fossa. In the large majority of cases the facial weakness corresponds to the side of the tumour, though in some it is the opposite facial muscles that have been weak.

Auditory Nerves.—In addition to the deafness which results from pressure on the auditory nerves, a similar result may be the outcome of pressure on the auditory tracts, and Dercum further suggests that in some cases it is due to pressure on the posterior corporo quadrigemina and internal geniculate bodies through which the auditory tracts are supposed to pass in their course to the brain. The deafness even when bilateral may be more marked on one side than on the other, and is commonly unilateral, in which case it corresponds to the side of the tumour.

Hypoglossal.—Paralysis of one side of the tongue is not common, and when met with is usually slight, and as a rule corresponds to the side on which the tumour is situated.

Speech.—Articulation is imperfect in a fair proportion of cases of cerebellar tumour, and the nature of the defect suggests that it is allied to the other ataxic disturbances that characterise cerebellar lesions; but pressure on the pons and medulla may, of course, account for the articulatory defects in some patients.

Deglutition.—Difficulty of swallowing is sometimes a symptom; usually under circumstances which make it probable that secondary pressure effects are responsible for the defect, though it may possibly be an ataxic difficulty in some cases.

Psychical State.—The mental hebetude which is so common in patients the subject of intracranial tumour in general, is met with in cases of cerebellar tumour, while in other cases the mental condition is one of irritability; but there is no clinical evidence to support the view of those that regard the hemispheres of the cerebellum as concerned with psychical functions.

State of Nutrition.—Patients the subject of cerebellar tumour are not more prone to waste than when the growth is situated in some part of the cerebrum, so that Luciani's view that the cerebellum exerts a trophic influence on the tissues of the body is not borne out by this, or indeed by any form of disease of the organ in man.

Sexual Functions.—Disturbances of these functions do not occur with any degree of frequency sufficient to justify the view held by Serres that the cerebellum is specially related to the sexual functions.

Atrophy.—The cerebellum may be atrophied as a whole and practically to extinction, while in other cases the change, though general, is not so pronounced. Both lateral lobes may be affected, the middle lobe alone remaining normal, or there may be only atrophy of one lateral lobe, a condition that is most commonly consecutive to a long-standing destructive lesion of the opposite cerebral hemisphere. Another cause of atrophy of the cerebellum is arrest of development, while a third is meningitis or some form of encephalitis. Hypertrophy of the cerebellum has generally been met with in imbeciles.

In some of the cases of cerebellar atrophy symptoms date from birth, while in others they follow definitely on an acute cerebral affection, prior to which no abnormality has been evident in the child. Nearly all the cases are mentally deficient; the impairment of intellect is slight in some, but pronounced in others, and the congenital cases may be idiots from birth. Dementia, melancholia, and maniacal attacks have been met with in a few cases. There is usually some abnormality of speech, though in some it may be quite normal. The particular form of defect and the degree of impairment varies; it is commonly slow, and may be drawling, hesitating, or jerky. Epilepsy is comparatively common in the subjects of cerebellar atrophy, though in many of the cases there are no convulsive attacks of any kind. Tonic spasm of the neck muscles is a symptom in some cases. In the majority of instances the patients have been able to sit or stand, but in some there has been an inability to sit upright, and a tendency to fall forward or backward on standing. It is exceptional for these cases to present any motor weakness of the limbs, and when present the legs have been usually affected. Movements may, nevertheless, be impaired in consequence of a variable degree of ataxy present, which may affect all the limbs or the arms alone, and, though bilateral, it may be more pronounced on one side than on the other. The movements may be uncertain and slow or jerky and irregular, or there may be tremor of the limbs of a more general character, the head and trunk may oscillate when the patient sits up, or the whole body may sway on standing. Others of the cases have shown no sign of ataxic disturbance, all movements being performed rapidly and with precision. The gait is usually reeling or staggering, and so great may be the disturbance that progression is scarcely possible; in a few cases, however, the gait has presented no abnormal features. Defects in the sphere of one or other of the cranial nerves may be present, but are rare. Among defects of the kind that have been met with are optic atrophy, dilatation of one pupil, strabismus, partial unilateral ptosis, and in a case with symptoms resembling those of disseminate sclerosis there was nystagmus. Weakness of the facial muscles on one or both sides has been noted, as has deviation of the tongue to one side on protrusion. The only other noteworthy defect in the province of the cranial nerves is difficulty in swallowing, which has been a symptom in a few of these cases. Cutaneous sensibility is unaffected, and it is exceptional to meet with any defect of muscle sense. Nothing pathognomonic has been established with regard to the knee-jerks; they have been normally preserved, diminished, or exaggerated in different cases. Sexual excitement has been a symptom in a few of the patients.

Cerebellar Heredo-Ataxy.—This affection may most appropriately be considered next. The disease was first described by Marie under the title Heredo-Ataxie Cérébelleuse, but its nosological place among diseases has not yet been definitely determined owing to the small number of cases that have come to autopsy. The affection is closely allied to Friedreich's disease; both affect the cerebello-spinal system, but there is reason to believe that that now under consideration begins at the cerebellar end of the system, while Friedreich's ataxy begins at the spinal end.

Etiology.—The principal fact in the etiology of the affection is its tendency to attack several members of the same family. The age of onset is, as a rule, after twenty. Practically nothing is known of any exciting cause, and though tubercle is not uncommon in such families, and alcoholism in a parent has been observed, it is doubtful if any significance is to be attached to these.

Morbid Anatomy and Pathology.—The number of cases from which we have to form our conception of the morbid anatomy and pathology of the disease are few, and in these the changes have not been uniformly the same. In one case there was widespread atrophy of the nervous system including the cerebellum; in a second there was degeneration of the spinal cord as well as of the cerebellum; in a third the cerebellum was atrophied but no change was detected in the spinal cord by the methods of examination then in vogue; and in a fourth there was no circumscribed lesion of the cerebellum, and only a slight decrease in the number of Purkinje's cells, while there was diffuse deterioration of the afferent cerebellar system, especially of the direct cerebellar tracts. The essential feature of the lesions appears to be a simple degeneration of the nerve elements without sclerosis or evidence of past meningitis. The resemblance of the leading symptoms to those caused by experimental and other lesions of the cerebellum have been regarded as evidence that this organ is the seat of the disease; but from what has been said of the morbid anatomy of the affection it is clear that this belief is not strictly borne out.

Symptoms.—The prominent symptoms are cerebellar in type. *Disturbance of equilibration* is evidenced by unsteadiness on standing which is not increased when the eyes are closed, and a reeling gait, though true inco-ordination of the lower extremities may be present especially in an advanced stage of the disease. The head participates in the unsteadiness, while the amount of this defect in the upper extremities varies with the stage of the disease; at first it is only an uncertainty, and then a tremor on voluntary movement as the hand nears the object aimed at, though later much coarser tremor is observed. Spontaneous choreiform-like movements occur in all parts of the body. True *motor paralysis* does not occur, but another motor defect met with in these cases is an inability to relax muscles that have been contracted, so that an object may be retained in the grasp longer than is dictated by the will. So, too, there is a tendency to associated *over-action of the facial muscles*, as evidenced by wrinkling of the forehead, wide opening of the eyes, exaggeration of the naso-labial fold and the like, when the patient speaks or performs any other facial movement. Apart from this the expression is dull and heavy. Intelligence is sometimes blunted and memory impaired, while irritability may be a feature in some of the cases. They speak in a monotonous way with a guttural voice, which may, however, change somewhat suddenly to a higher pitch, and words and syllables are emitted in a jerky, explosive manner. The ocular phenomena met with are important from a diagnostic standpoint. *Amblyopia* consequent on optic atrophy is constant. There is instability of globes, so that on fixation irregular, jerky movements are evoked similar to those that are observed in the limbs. *Ocular palsies* have also been present in some cases. Sensory phenomena are not prominent, though in some vertigo has been marked, so, too, headache, vague pains about the body, or shooting in character in the legs, and numbness have all been noted; but there is no blunting of cutaneous sensibility, and muscular sense is normal. The tendon jerks are normal or exaggerated. Difficulties of deglutition and micturition have been present in some cases.

The chief clinical distinctions between this affection and Friedreich's ataxy are to be found in the usual onset of the disease after the age of twenty, the presence of optic atrophy, the increase of the knee-jerks, and the absence of deformities such as scoliosis and talipes, for in Friedreich's disease the symptoms as a rule commence much earlier, optic atrophy does not occur, the knee-jerks are abolished, and spinal curvature and pes cavus are prominent features.

Diagnosis of Cerebellar Affections.—The points on which we rely as evidence that the cerebellum is the seat of disease have been sufficiently indicated in the accounts of the symptomatology of the different affections to which the organ is prone, and therefore need not be recapitulated. The considerations which aid us in the diagnosis of the nature of the disease do not differ from those on which we rely when other parts of the nervous system are affected.

Prognosis.—Hæmorrhage is commonly fatal, while thrombosis is of much less serious import, except in those cases in which the vertebral is occluded. According to Preston the average duration of life in cerebellar tumours is two years. The close proximity of the organ to the pons and medulla naturally allows of serious pressure on these structures, so that sudden death from failure of respiration is not an uncommon event. On the other hand, remissions may occur in the symptoms, and such patients may live for years, though blind. Atrophy of the cerebellum is compatible with comparatively long life.

Treatment.—The fact that the morbid condition is in the cerebellum does not make it necessary that the methods adopted in its treatment should be different from those employed when similar diseases attack other parts of the central nervous system.

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Cerebro-Spinal Meningitis. *See* Meningitis.

Cerebro-Spinal Rhinorrhœa. *See* Nose.

Cerebrum. *See* Brain.

Charcot's Disease. *See* Joints.

Cheek, Fissure of.—*Definition.*—A malformation of the lateral portion of the face, consisting in a cleft affecting the soft parts, or more rarely invading also the bones, arising during the embryonic period of antenatal life, and caused by delayed or irregular closure of the fissures between the fronto-nasal and superior maxillary and the mandibular processes.

Varieties.—From the anatomical as well as from the pathological and embryological standpoints fissures of the cheek may be divided into three kinds: (1) fronto-maxillary, (2) commissural, and (3) anomalous.

The *fronto-maxillary*, ascending, oblique, or naso-genal fissure begins at a point in the upper lip just outside the common site of hare-lip, and passes upwards outside the nostril towards the inner angle of the orbit; it may there produce coloboma of the lower eyelid, and it may, in exceptional cases, extend to the outer angle of the orbit and even on to the temple. It is unilateral or bilateral, simple or complicated; it varies in width from a narrow cleft to a gaping opening; it usually has a red floor and red irregular margins, but it may at one or more places present a cicatricial appearance (antenatal partial cure ?). When the underlying skeleton is also fissured the line of cleavage does not exactly follow the suture uniting the upper maxilla to the bones of the nose; generally the intermaxilla carrying the incisor teeth is internal to the fissure, but occasionally a precanine incisor is found external to it, a peculiarity not yet satisfactorily explained. The complications are the presence of other facial fissures (hare-lip, commissural cleft of the same or of the opposite side), deformity of the eye or eyelids, cerebral hernias and hydrocephalus, and the more distinctly teratological conditions of anencephaly, exencephaly, and adhesion of the placenta or amniotic membrane to the face or brain. It is very rare in animals (mammals).

The *commissural*, intermaxillary, or transverse fissure of the cheek (macrostoma, hiatus buccalis congenitus, commissural hare-lip, or coloboma of the cheek) differs in direction from the fronto-maxillary variety; it passes from the buccal commissure backwards, with an inclination upwards towards the masseter, and even when it proceeds further it rarely invades either the orbit or the external ear. It may be unilateral or bilateral, simple or complicated, and of great or little extent. When it is bilateral and extensive the mouth is converted into an opening reaching from one ear to the other. In the less marked degree the defect lays bare one or two molar teeth at the angle of the mouth. The opening of Stenon's duct can be seen in the upper margin of the cleft. The zygomatic arch may be cleft as well as the soft parts, and the upper maxilla may be small or deformed. Although it may occur as a solitary malformation, it is more common to find it associated with such anomalies as pre-auricular appendages (in eighty-seven cases of pre-auricular appendage collected from literature sixteen showed also commissural fissure, *v. Teratologia*, ii. p. 31; 1895), microcephaly, or hemi-atrophy of face or cranium, fronto-maxillary fissure, deformity of the external ear, cleft palate, macroglossa, and exencephaly. In animals (lamb, calf, pig) it is, on account of its extent, a grave condition, for the mouth may communicate with the tympanic cavity; it is called schistocephalus megalostomus.

Anomalous fissures of the cheek which do not follow the lines of the natural embryonic clefts, but which pass, for instance, from the angle of the mouth to the inner end of the orbit, or obliquely across the face from one side to the other, have been put on record. It is probable that they are usually the result of an amniotic adhesion, the other two varieties being due to simple pressure from an imperfectly developed amnion.

Symptomatology.—In addition to the obvious deformity caused by fissures of the cheek the symptoms of the commissural variety in particular consist in imperfect closure of the mouth, dribbling of saliva, and difficulty in mastication and articulation. Infants exhibiting this deformity are often small, weakly, and poorly nourished, and may be defective in intelligence.

Treatment has almost invariably consisted in the closure of the fissure by paring and uniting by sutures its margins. Generally the operation has been quite successful.

Cheloid. *See* CICATRICES.

Chest, Clinical Investigation of the.—In the clinical investigation of the chest the observer may gain much valuable information by a general survey of the patient, the appearance, expression, attitude, etc., before turning to the direct investigation of any cardiac or pulmonary derangement which may be suspected (Physiognomy). Yet the physiological functions and pathological alterations of cardiac and pulmonary organs are so intimately associated that it is impossible for an observer to differentiate how much of the patient's altered appearance and attitude may be due to disturbance of the functions of respiration and how much to disturbance of the circulatory organs, unless a careful physical examination of the chest be made, fully investigating the condition of the lungs and heart.

In examining the chest it is necessary to have the patient as far as possible at rest, with the limbs and trunk symmetrical and the light falling equally upon the parts under examination.

INSPECTION.—*FORM OF CHEST.*—*Normal.*—It is not common to meet the ideal typical chest, with a well-developed almost symmetrical bony frame, well clothed with muscle, fairly rounded on the anterior aspect, the interspaces visible below but not above, the obliquity of the ribs increasing from above downwards, and the epigastric angle nearly a right angle. Many alterations are met which cannot justly be considered pathological, thus as the result of occupation, *e.g.* the depression at the lower end of the sternum found in shoemakers. During health the respiratory and circulatory movements are accompanied by ceaseless changes in the outlines of the chest. With full inspiration, full expansion of the chest, the axes of the ellipse become approximately equal and the horizontal outline becomes more circular. The framework of the chest is shortened from above downwards, the ribs are raised and approach the horizontal plane, the intercostal spaces are narrow anteriorly, the epigastric angle is wide, the sternum is carried forwards and upwards, the shoulders are raised, and the scapulæ are closely applied to the chest-wall. Such is the chest of full inspiration. With full expiration the transverse axis of the ellipse markedly exceeds the antero-posterior, the ribs are depressed, the framework of the chest is lengthened from above downwards, the intercostal spaces are wide anteriorly, the sternum passes downwards and backwards, the shoulders are low, and the scapular angles project somewhat from the chest-wall. Such is the chest of expiration. While these changes in the outline of the chest are constantly going on in health, in disease the same changes occur in an exaggerated degree. In disease and deformity there may be an increase or diminution in the length or height of the bony framework, an alteration in the horizontal position of the ribs leading to an alteration in the axes of the ellipse, an increase or diminution in the width of the intercostal spaces, in the size of the epigastric angle, in the position and direction of the sternum and spine, in the height of the shoulders and the position of the scapulæ, and all these factors have a definite relation to the outline of the horizontal plane of the chest, and it is by the study of the outline of the horizontal plane that the observer will get the best idea of the alterations in the chest in disease and deformity. The outline of the horizontal plane of the chest is best ascertained by means of the cyrtometer. The best idea of the shape of the chest can be got by taking the outline at a given point with the cyrtometer. Various elaborate instruments are to be had, but a very good cyrtometer can be made with two bars of malleable metal connected posteriorly by means of a rubber hinge. The two bars, being moulded round the chest, and their point of intersection marked, they can then be detached and placed upon paper and an accurate tracing obtained. The outline of the adult chest is seldom symmetrical. Employments which cause individuals to assume certain positions or to use a certain set of muscles constantly are very prone to cause variations in the shape of the chest, without these variations having any direct pathological significance. The circumference of the right side is, as a rule, larger than the circumference of the left; in a small number of cases the left is the larger. In the adult the lateral diameter exceeds the antero-posterior, in the child the chest is more circular in form, the two diameters being about equal.

(1) *Alterations in Size and Shape, etc.*—In *emphysema* the chest presents a characteristic appearance, the neck appears shortened, the shoulders elevated, and the back rounded; there is a bilateral increase of the chest, the outline tending to assume the circular form seen in childhood. The chest is often described as "barrel-shaped." In the "*alar*" or "*phthisical* chest" we find sloping shoulders, prominent thyroid cartilage, deep hollow over the episternal notch, and prominent scapulæ. Here the antero-posterior diameter in the upper two-thirds is much shortened, and instead of a convexity there is usually flattening. The ribs tend to slope downwards instead of coming forward in a normal curve.

Unilateral increase of a general character is met with in effusion into the pleura. Localised increase may arise from acute croupous pneumonia, tumours of the lung, and mediastinal growth. Marked increase over the lower and right side of the chest should suggest the possibility of some hepatic affection, while on the left side the spleen should be remembered. In cardiac enlargement, especially when the enlargement has developed during childhood, there may be marked bulging or increase in the precordial region; the bulging from aortic aneurism and pericardial effusion should also be borne in mind. The deformities of the chest (*q.v.*) may also cause bulging, especially the apparent unilateral alteration due to spinal curvature.

Unilateral diminution may result from chronic fibroid phthisis, or from old-standing pleurisy or empyema. If the fluid in the pleural cavity be not evacuated before changes have taken place in the lung and pleura, the corresponding side of the chest-wall becomes retracted, secondary to a collapse of the lung. *Local flattening* may be noticed below the clavicle as a result of diminished functional activity of the upper lobes, which is usually associated with some organic lesions of a tuberculous nature. In children depression of the chest-wall may follow collapse or chronic interstitial pneumonia.

Before concluding the inspection of the chest the presence or absence of enlarged or pulsatory blood-vessels at the base of the neck should be observed. In emphysema and chronic bronchitis the vessels are distended, and a pulsation in the jugular veins is met with in cases of tricuspid regurgitation. Pressure on the superior vena cava and innominate veins from aneurising or intrathoracic new growth produces tortuosity of the veins above the clavicles and the engorgement of the veins of the trunk and limbs.

The Movements—1. *Lungs.*—In health these are respiratory and circulatory. The points to note in regard to the respiratory movements are (i.) *The rate of respiration.* The average of this is 14 to 18 per minute in the adult, in the child more rapid, about 40 per minute at birth, but gradually slowing as age advances. In health the ratio between heart-beats and respiratory movements is 4 to 1.

Dyspnœa, or difficulty in breathing, is a common and distressing condition in chest affections. Any cause which prevents the proper oxygination of the blood in the lungs will produce the subjective feeling of difficulty of breathing, and the objective signs of increased respiratory effort. The error may be in the respiratory tract, sufficient oxygen not reaching the pulmonary capillaries, in an insufficient flow of blood through the lungs, as in valvular disease of the heart, in the blood itself being unable to take up sufficient oxygen for the needs of the body, as in advanced anæmia. Dyspnœa may thus be both *inspiratory* and *expiratory*. It may be for the most part expiratory, as in emphysema, when the lung has lost its elastic power, or when a tumour obstructs the glottis during expiration. Inspiratory dyspnœa is seen when there is obstruction to the entrance of air at the glottis, as in membranous laryngitis, spasm, paralysis of the dilator muscles of the glottis, etc. When from difficulty of breathing the patient assumes the upright position the condition is termed orthopnœa, the position being assumed to give the extraordinary muscles of respiration the fullest possible play.

2. The *rhythm* of the respiratory movements is in health perfectly regular; in disease this regularity may disappear. A peculiar type of irregularity, known as *Cheyne-Stokes' respiration*, may be met with in advanced cardiac and renal disease and in cerebral affections. In health the relative duration of inspiration and expiration is in the ratio of 5:6. In disease this proportion may be lost. If there be an obstruction to the entrance of air into the chest, inspiration will be prolonged. There is marked prolongation of expiration when there is difficulty in expelling the air from the chest, as in asthma and emphysema.

3. The *type of the respiratory movement* differs in the two sexes. In the male it is abdomino-thoracic, in the female thoracico-abdominal. In disease these types may be altered. When the action of the diaphragm and abdominal muscles is interfered with, as in abdominal tumours, ascites, pain in the abdomen, the movements may be purely thoracic; when there is marked pleural pain the intercostal muscles may be more or less fixed, and the breathing be abdominal. Pure abdominal breathing is seen in its fully developed form in paralysis of the intercostal muscles. Diminished local expansion is seen when one lung, or part of a lung, becomes functionless, as in pleurisy with effusion, pneumonia, phthisis.

4. The *extent of the chest movement* has finally to be observed. In the characteristic "alar" and "barrel-shaped chest," previously described, the movements are very slight. Deficient expansion or inspiration is an important diagnostic sign. When one side of the chest moves more markedly than the other we may find a pneumonia, pleurisy, or tuberculous consolidation in the motionless side.

The Circulatory Movements.—The *apex beat*, a gentle pulsation visible and palpable in the fifth interspace internal to the mammary line, extending a single interspace vertically, laterally about one inch, regular in rhythm, and systolic in time, is in health the only cardiac movement visible in the chest. In disease it may be displaced from its normal position, the whole heart being displaced, as in pleurisy with effusion, when the heart is pushed to one or other side; in ascites or abdominal tumour, when it is pushed upwards; in emphysema and mediastinal tumour, when it may be pushed downwards. By far the most common cause of dislocation of the apex beat is, however, to be found in the heart itself. Marked displacement downwards and outwards is seen in hypertrophy and dilatation of the left ventricle, as in aortic disease; in hypertrophy and dilatation of the right ventricle the displacement is more outwards, less downwards; in pericardial effusion there is an upward and slightly outward displacement. Besides the apex beat, other pulsations may be visible, thus in the *third, fourth*, and *fifth interspaces* to the left of the sternum when there is hypertrophy of the right ventricle, or when the chest-wall is thin and the lung retracted, as in fibroid phthisis, the pulsation of a normal right ventricle may be visible. Pulsation at the *second* and *third right costal cartilages* may be seen in aneurism of the aorta. In retraction of the lung the pulsation of the conus arteriosus of the pulmonary artery may be visible. The observer notes then the pulsation, its position, extent, character, and time in the cardiac cycle, and the presence or absence of abnormal pulsation.

LUNGS.—*Palpation of the Chest-wall.*—The extent of the chest movements which have been observed under inspection should be verified by palpation, requesting the patient to take a deep breath. The expansion of the two apices, the two infra-clavicular regions, the axillary regions, and the bases of the lung should be in turn carefully investigated. Areas of superficial tenderness may be detected during palpation, and should be carefully noted. The state of the intercostal spaces should be noted, and the presence of various accompaniments, such as friction fremitus, râles, etc., may also be determined. The *vocal fremitus*, or the ability of the thoracic viscera and chest-wall to transmit vibrations produced by the voice, must then be determined. This is subject to great variation consistent with health, being more marked in men than in women and children, and is diminished when the chest-wall is well covered with muscles and fat. Further, the vocal fremitus is greater on the *right side* than on the left, owing to the relative positions of the right and left main bronchi. The diseases causing a *decrease of vocal fremitus* are pleural effusion, great pleural thickening, pneumothorax, and any condition which causes occlusion of a large bronchus, such as a tumour or copious secretion of mucus. *Increase of the vocal fremitus* is met with in consolidation of the lung (pneumonia and tubercle) or over a cavity surrounded by consolidated lung or a tumour in the thorax intimately connected with a bronchus.

HEART.—Palpation of the chest-wall will also give information as to the position and character of the cardiac pulsation, and thus confirms what has already been observed as to the position of the apex beat and the presence or absence of abnormal pulsation.

The cardiac pulsation may be accompanied by a thrill—a gentle vibratile sensation which is felt on placing the hand over the region of the heart. A thrill is produced in the same way as a murmur from fluid veins being set up within the blood-stream, the resulting vibrations being sufficiently ample to pass through the chest-wall and be perceptible to the hand of the observer. If a thrill is present note its time in the cardiac cycle and its position and character. A thrill presystolic in time (the most common variety) is usually perceptible in mitral stenosis. Systolic thrills, as in mitral regurgitation or aortic stenosis, are fairly frequent, diastolic exceedingly rare.

In percussion of the lungs the observer begins above the clavicles and passes down the chest in the mammary line, laying the pleximeter finger along the interspace or rib, and not across. Compare corresponding points of the chest-wall on the two sides, for if this be not done, the first degree of dulness, comparative dulness, may easily be missed. In percussing in the mammary region percuss outside the mammary line; internal to the mammary line on the left side, the sound will be altered by the underlying heart. When percussing posteriorly strong percussion must be used, except at the lower margins of the lungs. The patient should be bending slightly forward with the arms symmetrical and the head straight, for if the head be not straight, differing tension of the muscles over the apex will give rise to an alteration in the percussion sound.

The normal thoracic percussion sound got on striking over a thick layer of healthy resonant lung may be described as an ample percussion sound of considerable duration, low in pitch, and of a clear tone. In disease the reasonance of the sound may be increased; the sound may be hyper-resonant. Slight hyper-resonance is got in emphysema, more marked on percussing over relaxed lung, as above a pleural effusion, or where exudation and air containing tissue are intimately mixed, as in œdema, early pneumonia, etc. Marked hyper-resonance is met in pneumothorax, pneumopericardium. The resonance of the sound may be diminished—the sound is dull. Comparative dulness is got where there is some pleural thickening, in early phthisis, etc.; dulness where the consolidation is more advanced, as in pneumonia; absolute dulness is found typically in effusion into the pleura. In some cases the percussion sound may have a special quality, as the crack-pot sound, the amphoric sound, the bell sound.

Auscultation.—Next auscultate the lungs. Note the inspiratory and expiratory phase of the breath sound, their relative duration, their pitch and intensity, and the presence or absence of a pause between them, and thus deduce the type of the breath sound.

The *normal type of breath sound* heard all over the lung except at the interscapular region is vesicular. Inspiration is a gentle continuous rushing sound; expiration a thinner, fainter sound, which at times may not be audible; inspiration is three times as long as, and passes directly into, expiration. In disease this vesicular murmur becomes altered. The breath sound may be harsh, as heard in the normal child's chest, or over the healthy lung when the other is rendered functionless, as in pneumonia or pleurisy with effusion. *Harsh vesicular breathing* with prolonged expiration is heard in slight consolidation of the lung, as in tuberculosis, the audible part of expiration being prolonged. Wavy, jerky, or interrupted vesicular breathing may be nervous in origin, but it usually results from delay in the entrance of air into the pulmonary bronchi through catarrh giving rise to obstruction, and when localised is to be regarded with suspicion. Clinically it is found in early tuberculous infiltration, bronchitis. *Faintness or absence of the breath sounds* may be present if the sound vibrations produced at the larynx and upper part of the respiratory tract are cut off by obstruction of a bronchus, effusion into the pleural sac, thickening of the pleura, etc.

When in disease the sound vibrations produced at the naso-pharynx, pharynx, and larynx are not damped down by passing through spongy air-containing lung, the sound over the chest has the same characters as at its point of production, and, being similar to the sound heard on listening over a bronchus, is called *bronchial.* Inspiration and expiration are about equal in length, they are separated by a distinct pause and have a blowing quality. Three kinds of bronchial breath sounds are recognised, distinguished according to their pitch. High pitched or tubular breathing; medium pitched or simple bronchial; low pitched or cavernous. The simple or medium pitched variety is heard where the lung is a better conductor of sound, as in consolidation in phthisis. The high pitched variety, more intense and harsh in character, is found where consolidation is more complete, as in croupous pneumonia. The low pitched or cavernous variety differs from the other varieties in its pitch and in having a distinctly hollow quality. It is heard over cavities in the lung, phthisical or bronchiectatic.

Broncho-vesicular Breathing.—The type of breath sound heard in health in the interscapular region and over the manubrium sterni possesses some of the characters of bronchial and some of the characters of vesicular breathing. Pathologically it indicates some slight increase in the conducting power of the lung, as in early tuberculous infiltration.

Amphoric breathing, a rare variety, has a hollow whistling character. Like bronchial, it may be high, medium, low pitched. It may be heard over a pneumothorax where there is free communication between the pleura and a bronchus, or over smooth-walled cavities having free communication with a bronchus.

Accompaniments.—Having determined the type of the breath sounds, notice the presence or absence of accompaniments. If present they may be extra-pulmonary, such as friction. Intrapulmonary accompaniments may be dry râles or rhonci, snoring, cooing, whistling, or creaking sounds due to an alteration in the lumen of a bronchial tube, as in asthma or bronchitis, or there may be moist râles or crepitations, sounds which convey to the ear the idea of the bursting of small bubbles or the crackling of hair rubbed between the fingers.

Examine next the *vocal resonance,* asking the patient to repeat ninety-nine, or one, one, one. In health the sound vibrations are conducted to the stethoscope altered by their passage down the column of air in the respiratory tract and through the chest-wall. In disease when the lung is consolidated, as in phthisis, pneumonia, the sound is increased. When the sound is markedly increased, it is called *bronchophony.* The sound vibrations are diminished or lost when there is diminished facilities for their conduction, as when a bronchus is plugged, when there is thickening of the pleura, effusion into the pleura, œdema of the chest-wall, etc.

Under certain conditions the whispered voice is well heard, as if spoken into the end of the stethoscope. This is known as *whispering pectoriloquy* (see article "Lungs (Phthisis)").

Percussion of Heart.—Turning next to the *percussion of the heart,* two areas of dulness are recognised: the superficial or area of absolute dulness, the deep or area of relative dulness. The *superficial cardiac dulness* corresponds to that layer of the heart which lies uncovered by lung, having a truncated pyramidal shape, with its right border along the left border of the sternum, its upper boundary at the fourth costal cartilage, its left border about the parasternal line. The size of the area varies with the state of expansion of the lung. In enlargement of the heart it is increased, in emphysema it is diminished or absent, the over-expanded lung occupying the area. The area of *deep cardiac dulness* is of much greater importance to the clinician. Using strong percussion percussing from without inwards along the third and fourth right interspaces, the right border of the heart is found about two inches to the right of the mid-sternal line. The left border is next mapped out, percussing along the interspaces from without inwards, beginning outside the mammary line, where a clear thoracic percussion sound is obtainable. By marking in each interspace when the relative dulness is first reached and joining the points, the position of the left border of the heart can be obtained. In health it is usually *in* the mammary line, that is about 3¾ inches to the left of the mid-sternal line at the level of the fourth interspace. In percussing the deep cardiac dulness, much assistance will be got by taking into consideration the sense of resistance when carrying out the percussion. The cardiac dulness is found to be increased in cases of dilatation and hypertrophy and in dilatation. In hypertrophy and dilatation of the left side the increase in the area is downwards and outwards; in hypertrophy and dilatation of the right side the increased dulness is more in the transverse direction. In effusion into the pericardium the dulness takes the shape of the pericardial sac; it is somewhat pear-shaped, the stalk of the pear being upwards. It is sometimes, as in emphysema, impossible to map out the cardiac dulness with certainty. A good deal, however, will depend upon the skill of the observer. Accurate percussion of the heart requires much practice.

Auscultation.—Taking next the auscultation of the heart, the different cardiac areas are listened at in turn, beginning, as a rule, with the mitral, and the character of the sounds is noted. Are the sounds healthy? If not healthy, is the alteration quantitative? Are the sounds louder or fainter than in health? or is the alteration qualitative? Is there a murmur present? Quantitative alterations may be various; the first sounds may be short, sharp, and accentuated, as in dilatation; it may be doubled, a somewhat rare condition; it may be prolonged and of grave tone, as in the hypertrophy and dilatation of chronic kidney disease. The second sound may be loud and accentuated at either area; it may be faint, or it may be doubled.

Is the alteration in the sounds qualitative? Is a murmur present? If so, time the murmur in the cardiac cycle—systolic, diastolic, or presystolic (auriculo-systolic); ascertain its point of differential maximum intensity; its sound character, duration, and the direction of propagation.

Extra Cardiac Areas.—Having auscultated the heart, the extra cardiac areas are noticed, the upper part of the sternum, the roots of the neck, and the epigastric region.

Palpate the upper part of the sternum for any pulsation or thrill, and percuss to ascertain if there is any increased impairment of the percussion sound, as in aneurism or dilatation of the aorta. Notice the root of the neck for any swelling, undue pulsation, and for venous pulsation, as in tricuspid regurgitation. The epigastric region may show pulsation, tumour, etc., which may have important bearing upon the condition of the heart.

In many cases the simple clinical examination as sketched is insufficient.

Extra auscultation will frequently give valuable information. Note the cough, its character, frequency, etc.

Note the presence or absence of expectoration, its amount, reaction, appearance, naked eye and microscopic, etc.

In cases where effusion into the pleura is suspected, or where the nature of an existing effusion is in doubt, it is often advisable to explore with the needle of an exploring syringe or aspirator.

In cardiac cases useful information may be got from the sphygmograph, cardiograph, sphygmomanometer, or sphygmometer.

The Röntgen rays have been applied to the diagnosis of respiratory and cardiac disease, but so far there are a number of difficulties in their application.

Controlling evidence is of much importance in many diseases of the chest. Examination of the larynx is of the greatest importance in cases of early phthisis, suspected aneurism.

The condition of the blood, the red corpuscles, the leucocytes, and the hæmoglobin are all of importance in cardiac and respiratory disease. Thus the presence of anæmia may account for a cardiac murmur, which otherwise must have been considered organic; the presence of a leucocytosis may give confirmatory evidence of a deep-seated patch of pneumonia.

Chest, Deformities of.

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In describing deformities of the chest it is most convenient to consider them in relation to their causal factor. The deformity may be the result of a developmental error, or it may be acquired. If acquired, it is the result of disease of the parietes, of the thoracic organs, or from the effects of external mechanical causes.

(i.) Congenital Deformities of the Chest.—Congenital deformities of the chest are comparatively rare. Cleft sternum results from deficient union of the visceral layers in the embryo. If the fissure be marked, ectopia cordis results. Defects of the lateral or posterior thoracic wall generally arise as the result of amniotic adhesions, and protrusion of the lung may then take place.

(ii.) Deformities due to Alterations in the Parietes.—The deformities of the *spinal column* produce a deformity of the thorax, slow, progressive, and at times considerable. In *scoliosis or lateral curvature* of the spine, the curvature of the vertebral column modifies little by little the direction of the ribs. The curvature of the ribs is increased on the side of the vertebral convexity, diminished on the side of the vertebral concavity, and, as a consequence, there is a protrusion of the chest on the side of the spinal convexity and a depression on the side of the concavity, and on the side of the spinal concavity the ribs approach each other, and may even overlap. The horizontal outline of the chest assumes an ellipsoidal form with a posterior protrusion of the ribs on the side of the scoliotic convexity, and a projection of the anterior angle of the ribs on the opposite side. The half of the thorax corresponding to the convexity is diminished in capacity; the opposite half retains its sectional capacity, but is diminished in vertical measurement. The shoulder on the side of the scoliotic convexity is markedly elevated, the scapula is thrown out behind, its angle is elevated, and is carried out from the middle line. In a number of cases a very complicated deformity is produced as the result of curves of compensation in the vertebral column, but there is always a diminution in the capacity of the thorax on the side of the scoliotic convexity, and as a result of this the action of the heart is impeded from displacement, and increased work is thrown on the right side in carrying on the pulmonary circulation.

Kyphosis or backward arching of the spine produces a flattening of the sides of the chest, and an increase in the antero-posterior diameter of the chest. The sternum becomes curved about its middle, and there thus results an anterior convexity or arching, more rarely an anterior concavity of the chest. The infra-clavicular depressions are exaggerated, the inferior angles of the scapulæ project from the chest-wall in the "alar" fashion, the abdomen frequently assumes an anomalous anterior projection.

In *Pott's disease or angular curvature* of the spine the deformity varies with the position of the spinal curve. If the curve be in the upper dorsal region the thorax is flattened from before backwards, the antero-posterior diameter of the horizontal section is diminished. When the projection is in the inferior dorsal region the thorax is flattened transversely, the lateral diameter of the horizontal section is diminished, and the outline of the section assumes a circular form.

In *osteomalacia* the deformities of the chest are secondary to the incurvation of the spine and the softening of the ribs, and in advanced cases may be very complicated.

In *osteitis deformans* the spine, more or less ankylosed, is inclined forwards in a dorsal lordosis. The ribs are fixed posteriorly, the chest is flattened laterally. The respiration is impeded, and is almost purely diaphragmatic.

In *acromegaly* the chest is projected forwards, the antero-posterior diameter of the horizontal plane is increased, the lateral aspects flattened, and the lateral diameter diminished. The anterior projection is most marked in the lower sternal region, and thus the sternum lies obliquely with its anterior surface directed forwards and upwards. The sternum is thickened, widened, and elongated. The anterior surface shows a series of grooves. The angulus Ludovici is very prominent, the xiphisternum is elongated and ossified. The clavicles are enlarged and the extremities thickened. The ribs are massive, so increased in bulk that their edges may be almost in contact, obliterating the intercostal spaces; the costal cartilages, broadened and ossified, form a chaplet, which may pass beyond the plane of the anterior aspect of the sternum. The spine may show a pronounced dorsal kyphosis. The whole bony framework of the chest is hypertrophied and the respiration is impeded, being abdominal in type.

In *rickets* the costal cartilages are abnormally firm, while the ribs are softer than natural, especially at their enlarged growing ends. The deformity is characterised by the formation of a groove running down the chest-wall almost parallel to the direction of the sternum, situated outside the junction of the rib with its cartilage, extending down to the costal margin. There is an increase in the antero-posterior diameter of the sectional outline, the sternum being carried forwards, and a diminution in the transverse diameter. In addition to this alteration in the outline of the chest, there is an enlargement of the cartilaginous ends of the ribs, which results in the beaded appearance known as the "rickety rosary." The deformity is the result of the atmospheric pressure acting upon the softened ribs. When the diaphragm descends during inspiration the rickety softened ribs are unable to withstand the atmospheric pressure during the time the fresh supply of air is entering the lung. The chest-wall yields at its softest part, that is, immediately external to the enlarged growing ends of the ribs, and a sulcus forms down the sides of the chest, while the sternum is carried forwards by the unyielding costal cartilages. The rickety deformity may form without any actual impediment to the entrance of air into the chest, but is accentuated if any impediment such as a bronchial catarrh be superadded to the rickets. The deformity being due to an undue softness of the chest-wall, the solid organs subjacent may modify the deformity; thus the liver supports the lower chest-wall on the right side, while the heart may form a prominence on the left side.

In *syringomyelia* a boat-shaped hollow sometimes develops in the upper part of the thorax, the region below the level of the fifth rib being normal. The head is buried between the shoulders, and the sternum shows considerable obliquity and the shoulders are carried forwards. There is no functional disturbance.

(iii.) DEFORMITIES OF THE CHEST DUE TO MECHANICAL CAUSES.—Any more or less continuous alteration in position or increased pressure will affect the outline of the thorax and give rise to greater or less deformity. Elevation of one shoulder, usually the left, is met with in clerks who, on account of the position taken while writing, tend to have a slight lateral spinal curvature with elevation of the shoulder. Again, in persons whose occupation necessitates the carrying of heavy weights on one arm, the opposite shoulder takes up a higher position, and a slight deformity is induced. In some trades considerable deformity may result from pressure, as, for example, the well-recognised depression which occurs at the lower end of the sternum in shoemakers from the pressure of the "last." In the developing chest of young boys a projection of one or more costal cartilages may result from lateral compression in athletic exercises. The cartilage, being soft when it is compressed between the rib and the sternum, bends, and an angular projection forms. In the female, mechanical modification may result from tight corsets. The lower ribs are forced inwards and imprint their traces on the viscera, the massed intestine presses upon the pelvic organs, the descent of the diaphragm is limited, and the respiration becomes thoracic in type; the respiratory capacity of the lower part of the chest is diminished, and the epigastric angle is reduced. The pressure results in enteroptysis, or else, if dislocation of the different organs be not produced, an indelible impression remains with an alteration in the shape of the organ.

(iv.) DEFORMITIES DUE TO ABNORMAL CONDITIONS OF THE THORACIC ORGANS.—The framework of the chest undergoing, as it does, ceaseless temporary changes in outline during the respiratory and circulatory movements, the shape of the outline must largely depend upon the condition of the contained viscera. Any alteration taking place in the thoracic viscera must to a greater or less extent tell upon the parietes. The development of the chest must be dependent upon the development of the contained organs, and any want of respiratory capacity will tell in the most marked manner upon the form and outline of the chest. While thus departing from the normal the form may not amount to absolute deformity, but the abnormality is of importance to the physician as showing the existence of past or the probability of future lung mischief. Of such a type is the "alar" or "pterygoid" chest. In such individuals, as the result of improper surroundings and education during early life, there is deficient lung development, or it may be that there is a congenitally small lung capacity. As a consequence there is deficient development of the chest; it is shallow and narrow, there is an increased obliquity of the ribs which results in a proportional diminution of both the antero-posterior and transverse diameters, the increased obliquity of the ribs results in a drooping and forward inclination of the shoulders, the upper part of the scapula is thus carried forwards and the angle tilted backwards from the ribs, giving the chest the peculiar "alar" appearance. While the increased obliquity of the ribs results in increase in the vertical length of the bony framework, the vertical capacity of the chest is really diminished by an increase in the height of the diaphragm.

The flat chest, the type of the tuberculous, shows a marked and distinctive deformity. There is a loss in the normal anterior rounding of the chest, there is a diminution in the antero-posterior diameter of the sectional outline in the marked form, the cartilages of the true ribs having a diminished anterior curve, in some cases the sternum even being depressed. In the flat chest, as distinguished from the alar, there may be no increased obliquity of the ribs. Both alar and flat chests are essentially deformities the result of deficient lung capacity, which is said by some to be congenital, but may also be the result of deficient pulmonary development arising from adverse circumstances and surroundings.

The *pigeon* chest is characterised by an alteration of the almost circular sectional outline of the childish chest to a triangular form. There is an increase in the antero-posterior diameter of the chest due essentially to a straightening of the true ribs in front of their angles, and the sternum is thus carried forwards. The *transversely constricted* chest is a very common form of deformity. The chest shows a depression or groove which, commencing at the level of the xiphisternum, passes outwards and downwards till it shades away towards the mid-axillary line. The transversely constricted and pigeon deformities have essentially the same causal factor—an impediment to the entrance of air into the lung during the developmental period of life, while the chest-wall is soft and yielding. Given an affection of the respiratory tract which will interfere with the free entrance of air, such as enlarged tonsils, bronchitis, whooping-cough, etc., and either deformity may result. The respiratory obstruction causes inspiratory dyspnœa with increased efforts at inspiration. The chest and lung are more easily expanded above than below, hence when the diaphragm descends and the ribs at the upper part of the chest are drawn up, the lungs not expanding fully at their lower part, there is a tendency to form a vacuum at the lower part of the chest, and the atmospheric pressure forces in the chest-wall and a sulcus is thus formed. The sulcus corresponds to the upper level of the abdominal organs which support the lower part of the chest-wall. The transversely constricted chest is thus developed. When the inspiratory dyspnœa has been more pronounced and more persistent, the deformity advances to the pigeon chest. Forced inspiration over-expands the upper thorax, protrusion of the sternum takes place, the atmospheric pressure forces in the lower part of the chest, producing the transverse sulcus and a bending back of the xiphisternum. From the altered position of the sternum the costal cartilages become straightened in front of their angles, and the greatest transverse diameter of the chest lies posterior to the normal position.

The *emphysematous chest* is characterised by an increase in all the diameters of the sectional outline of the chest—from the ellipse the outline assumes a circular form. Repeated and prolonged inspiratory efforts produce an overdistension of the lung; the lung becomes emphysematous. The lungs have become too voluminous to be accommodated within the fixed peripheræ of the chest when in an ellipsoidal form, the fixed peripheræ assume a circular outline to give a greater contained area for the enlarged lungs. The outline of the chest is circular, there is an increase in all the diameters, the sternum is arched, the shoulders are raised, and the spine is arched; the chest is fairly described by the epithet "barrel-shaped." The enlargement of the chest may be confined to the parts above the level of the xiphisternum, but frequently the whole chest is affected, in which case the epigastric angle is markedly enlarged.

Unilateral alteration in the shape of the chest may be seen in a variety of conditions affecting the lung or pleura.

A unilateral dilatation of the chest is rarely seen in the healthy side when disease impedes the action of the other lung. On the diseased it is markedly apparent by measurement or inspection in pleurisy with effusion, pneumothorax, and in cases of tumour of the lung. The intercostal spaces are obliterated, at times they may even bulge. Movement on the affected side is diminished, exaggerated on the sound side.

Diminution of one side of the chest may form a very striking deformity. It may follow upon chronic fibroid and destructive changes taking place in one lung, or upon pleurisy with effusion or empyema. In pleurisy or empyema, while the fluid is present, the lung is collapsed; if before the fluid be evacuated changes take place in the lung and pleura, preventing the expansion of the lung when the fluid ultimately becomes evacuated or absorbed, marked deformity results. The lung can no longer expand to fill up the affected side, the heart passes over towards the affected side, the opposite lung becomes emphysematous, and the atmospheric pressure forces in the chest-wall on the affected side. In such a condition there is marked deformity and diminution on the affected side, and the heart is displaced towards it. Marked deformity of a similar nature may result in chronic fibroid phthisis.

Local deformity of the chest-wall is frequently met. Local bulging or increase may be seen in circumscribed pleural effusions, in tumour of the lung, in hernia of the lung, in cardiac hypertrophy, in aneurysmal tumour.

Local contractions or diminution of the chest-wall due to intrathoracic disease is seen in phthisis even without the formation of a cavity, though it is more marked if a vomica be present.

Chest, Injuries of.

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SURGICAL AFFECTIONS OF THE CHEST-WALL

1. SOFT PARTS.—Concussion and contusion of the chest without evidence of injury to internal organs are of frequent occurrence, and in many cases lead to no serious results, but it must be borne in mind that an injury which appeared slight at first may later give rise to alarming symptoms. In concussion from the buffers of a railway carriage or the pressure of part of an ordinary carriage on the chest-wall very little may be observed at the time, and later extensive hæmorrhage from an injured internal organ may lead to serious complications and death. In adults this is oftener observed than in children, as their chests, being more yielding, bear concussions much better. The shock from slight concussions is often much greater than might be expected, because the influence produced by them varies greatly both in adults and children, and cases are on record where a slight blow on the chest has caused profound shock and sometimes death. It is therefore extremely important to treat all cases of concussion and contusion of the chest, even when slight, with the greatest

care, avoiding prolonged examination during the period of shock. Laying the patient flat in bed with warmth judiciously applied round him, using stimulants in small quantity and often repeated, with subcutaneous injections of strychnine and ether where the tendency to heart failure is great, will generally be sufficient in ordinary cases of shock. As very serious injuries internally may result from blows on the chest without any external wound or any fracture of the ribs, it is important to keep the patient at rest for eight or ten days, by which time it may be assumed that the dangerous period is past.

Ordinary wounds not involving penetration into the chest, such as cuts and stabs only passing through skin and muscle, occur frequently, and should always be treated with the same antiseptic precautions as wounds elsewhere. When of small size, after thorough cleansing, a simple collodion dressing may be sufficient, but where extensive, stitches are required and aseptic or antiseptic dressings, according to the opinion of the surgeon in charge. In lacerated wounds and in very slight wounds with a large amount of extravasated blood in the tissues special care ought to be taken in the cleansing process, as should suppuration take place it spreads with great rapidity in the cellular tissue of the chest. In all such cases, and where wounds involve the muscles, it is important to note that the fixing of the dressings is so arranged that the arm is bandaged to the side, thus securing rest to the injured muscles. At the end of ten days the parts are generally sufficiently healed to allow the arm free of the bandages, and passive motion is employed to prevent stiffness.

Wounds involving both the thorax and abdomen are comparatively common; the vault of the diaphragm reaches the level of the fifth, and it may be the fourth rib on the left side; the pleura lines practically the entire wall of the thorax; hence a penetrating instrument is liable to traverse the pleural cavity and enter the cavity of the peritoneum. The injury to the abdominal viscera may be the more serious element; the organs most likely to be injured are the stomach, liver, spleen, and kidney. Perforation of the diaphragm on the left side may result in one or other of the forms of diaphragmatic hernia (see "Diaphragm, Surgical Affections of").

2. THE RIBS AND STERNUM.—*Contusions* of the ribs are very common, and are caused by direct violence, producing frequently very persistent pain over the site of injury. If this pain does not yield to soothing fomentations with belladonna or laudanum for forty-eight hours, and thereafter the application of strips of ordinary sticking plaster applied for a week, a small fly blister over the site of pain is very frequently of great service.

Fractures occur generally from the fourth to the eighth ribs, the upper and lower ribs being more protected; the floating ribs yield more to injuries, and consequently are less often broken. The most frequent seat of fracture is usually at one or other end of the rib, and the ribs most frequently broken are the fifth and sixth. A rib may be broken in two places, or may simply be cracked. The diagnosis is not always easy, a simple fracture being most easily felt by placing the hand flat on the chest and directing the patient to take a long breath. Sometimes placing two fingers over the site of pain and pressing one inwards enables crepitation to be made out. It is a good plan to examine carefully each rib with the fingers, in order, if possible, to make out crepitation. Sometimes this is so difficult that it is not possible to be absolutely certain as to whether the rib is broken or not, but if the patient has intense pain at the end of inspiration or in turning suddenly in bed, and if his respiration is short and hurried, we are justified in concluding that he has had one or more ribs broken. Ribs are generally broken by direct violence, but a number of cases are on record where after violent sneezing or coughing one or more ribs have given way, and in the insane simply turning in bed has been known to fracture several ribs due to disease of the bone. Fractures of the ribs cause serious complications, with which we will deal later on when speaking of injuries to the viscera. The treatment of a simple fracture consists in strapping the injured side and applying a broad bandage round the chest, and where this gives comfort it may be continued; in certain cases, however, patients are easier without any bandage at all. The fracture is generally united in three weeks, and it is unnecessary to keep patients in bed in uncomplicated cases for longer than a few days. The *costal cartilages* are sometimes broken by direct violence, or a severe crush of the chest may lead to several costal cartilages giving way, and the same treatment is employed as for fracture of the ribs.

Dislocations of the ribs.—Separation of the head of a rib from its articulation with the spinal column is exceedingly rare, while dislocation of a costal cartilage from the sternum is very uncommon. Cases have been mentioned both of forward and backward displacement, the forward being easily reduced by pressure, the backward requiring pressure on the sternum while the patient takes a deep breath.

Sternum.—Fractures of the sternum may be transverse or oblique, and are caused either by direct or indirect violence. They occur between the manubrium and the gladiolus, or at the ensiform cartilage. The manubrium is generally displaced backwards and behind the gladiolus, and is reduced when possible by placing the patient on the back, putting the knee between the shoulders, and pressing the ribs and gladiolus downwards. Sometimes it is impossible to reduce the displacement, and unless the manubrium is giving rise to uncomfortable pressure symptoms, operative interference is unnecessary. The ensiform cartilage may be driven backwards towards the spine by direct violence, and from pressure on the stomach vomiting may become so persistent that an operation to replace the displaced cartilage becomes a necessity. It is well to bear in mind that in both fractured ribs and sternum the intercostal and internal mammary arteries have been injured and the patients have died from fatal hæmorrhage. Compound fractures of the ribs and sternum are dealt with in exactly the same way as compound fractures elsewhere. It is sometimes necessary to remove portions of rib in such circumstances, and the pleural cavity is as a rule penetrated, and therefore care must be taken to ensure careful drainage.

3. The Viscera.—Wounds of the lung lead to various symptoms, according to their extent, and may be produced by simple concussion, fractured ribs, stabs by knives, swords, and such-like weapons, or by gunshot wounds or any projectiles having sufficient force to penetrate the chest-wall. Hæmoptysis, emphysema, pneumothorax, hæmothorax, are the symptoms which occur soon after an accident, while some days after an injury to the viscera we may have pneumonia, abscess of the lung, bronchitis, gangrene, or hernia of the lung.

Hæmoptysis to a slight extent may occur in concussion or slight injury to the substance of the lung by a broken rib, and is frequently accompanied by *emphysema*, which consists in the air passing into the cellular tissue through the opening in the lung, and is diagnosed by placing the hand over the chest and feeling the crackling caused by the air in the cellular tissue. Where the wound in the lung is extensive, as by a sword thrust, the emphysema may extend over the whole body, and the patient may become unrecognisable.

This condition, however, is not very common. The treatment of moderate emphysema consists in strapping the chest in cases of fracture and dressing the wound antiseptically where it has occurred from a stab. Where, however, the emphysema is general, free incisions must be made into the cellular tissue to allow the air to escape, not forgetting the importance of the incisions on each side of the larynx, as the pressure in that region is so great as to threaten suffocation, and patients have died from neglect of this precaution.

Pneumothorax. — In some cases of injury to the lung, instead of emphysema occurring, the air rushes into the pleural cavity, compressing the lung and giving rise to the most distressing dyspnœa, which is relieved by passing a trocar and canula into the pleural cavity and allowing the air to escape, having previously of course used antiseptic treatment for the skin. After the air has escaped a rubber tube with a flange (to prevent its passage into the chest) is introduced and antiseptic dressings applied.

Hæmothorax.—In many cases in addition to air in the chest a large quantity of blood may exist from injury to the vessels in the lung. Where blood alone is extravasated the condition is called hæmothorax; where both air and blood are present it is called hæmo-pneumothorax. These cases are generally so serious that no interference is possible, and patients generally die from the extensive hæmorrhage. Venesection is recommended in cases of hæmorrhage in order to produce faintness and possible arrest of the bleeding. A certain number of cases recover from arrest of hæmorrhage and absorption of the fluid. The balance of surgical experience points to the importance of not interfering unless there is evidence of empyema. Where there is a wound into the chest with free hæmorrhage the question of opening up the wound, turning out the clots, looking for the bleeding vessel or vessels, and if possible securing them, must be considered and carried out in certain cases. Sometimes the hæmorrhage is so great on turning out the clots from the chest that it is better to stuff the cavity with gauze.

Traumatic hernia of the lung consists in the protrusion and often the strangulation of a portion of the lung through a wound in the chest-wall. It is usually met with in wounds which open into the pleural cavity without involving the lung itself. The wound in the chest-wall must be of a certain size to allow of the protrusion of the lung, and it must correspond in position with one of the borders of the lung or with the corner of one of its lobes. The hernia may take place as soon as the penetrating weapon is withdrawn, or it may not take place for many hours after the injury. Violent expiratory movements have a good deal to do with its production; part of the air expelled from the lung on the healthy side may be driven into the partially collapsed lung on the injured side, so that it becomes distended and may protrude at the wound. If, on the other hand, the lung itself is wounded, and especially if one of the bronchi has been opened into, the air simply escapes into the pleural cavity. In the first instance, it is easy to return the protruded portion of lung, but after a time it tends to become strangulated, congested, œdematous, and irreducible, and may finally become gangrenous and slough away. Healing is then quite satisfactory as a rule, the wound in the chest-wall is closed with scar tissue, and the lung remains firmly adherent to the parietes.

Bullet wounds of the lung, as met with in civil practice, are usually caused by shots from a revolver. They have little penetrating power, and rarely traverse the thorax as a whole; they may lodge in the lung, or in the posterior wall of the chest, often beneath the skin. The injury produced resembles that caused by any other penetrating instrument. The wound in the skin is small, and gives exit to a little ooze of blood. The features resulting from injury to the lung depend on its situation and extent; the outlying portions may be wounded without any definite symptoms; penetration of the root of the lung or its vicinity may result in pneumothorax, emphysema, hæmoptysis, hæmothorax, etc. The presence of the bullet, as a foreign body in the chest, does not appear to add to the gravity of the injury. The rule is not to be too eager to follow the course of a bullet in the chest, unless it is easily felt or is producing symptoms of irritation.

The large calibre and slow velocity of the rifle bullet of former days were attended with more extensive injury of the parietes, and were more likely to be complicated by the carrying in of portions of clothing, splinters of bone, etc., and the wound of the lung was often extensive, lacerated, and infected. With the modern rifle and bullet there is more likely to be a clean small hole right through the chest.

Wounds of the heart and pericardium are usually produced by pointed instruments or by bullets which penetrate the wall of the chest; there are certain rare cases on record in which a pointed foreign body in the œsophagus has penetrated the heart-wall.

The wound of the chest-wall is usually in the precordial region, and may appear to be of very little importance. The pericardium may alone be wounded, probably when it is on the stretch between its two attachments. The wall of the heart may be penetrated or perforated; the point of the weapon may be arrested in the myocardium of the ventricles, or may enter one or other of the cavities of the heart. The nature of the wound varies with the instrument causing it; the puncture of a needle is readily filled with clot; a stab with a knife may give rise to a wound which gapes; both ventricles may be penetrated; the apex of the heart may be cut off; a bullet may pass right through two or more cavities, or it may lodge in one of the latter; a bullet of high velocity may so raise the hydraulic pressure in the cavities of the heart that their walls may be extensively ruptured.

The pleura and lung are very commonly injured at the same time as the heart, especially the anterior reflection of the pleura on the left side. The great vessels of the chest may also be involved, *e.g.* aorta, vena cava, pulmonary vessels, azygos vein, etc. A wound of the heart may interfere with its functions in various ways; its action may be arrested altogether by the injury to its walls or to its valves, or by the influence of the injury on its nerve mechanism, so that the individual dies of syncope; accumulation of blood in the pericardium (hæmopericardium) is a very constant accompaniment of wounds of the heart; it may be derived from one of the coronary arteries or from one of the cavities; if it does not escape externally from the wound in the pericardium, it accumulates and presses directly on the heart, so that the latter may cease to beat; if it does escape through the wound in the pericardium, the patient may bleed to death.

If the pleura has also been wounded, the blood may pass into the pleural cavity and result in an extreme form of hæmothorax.

Should the patient survive, he may become the victim of septic complications, of which purulent pericarditis and pleurisy are the most important examples.

The symptoms associated with the lesions described are very variable and inconstant. The patient may present no evidences of serious injury; he may be able to stand, or even walk; more often there is a condition of syncope or collapse, which may be rapidly fatal; there may be external hæmorrhage sufficient to cause death in a few minutes, or there may be very little or none at all. The pulse is small, rapid, irregular, and intermittent; the heart sounds may be faint or inaudible, or they may be replaced by murmurs of very varied character; an increase in the area of the cardiac dulness may indicate hæmopericardium. Dyspnœa is very common, and may culminate in asphyxia; it may depend on the insufficiency of the pulmonary circulation resulting from the feeble and injured condition of the heart, or it may be due to complications such as hæmothorax and pneumothorax.

Delirium, convulsions, paralysis are occasionally observed; the latter one usually the result of cerebral embolism.

The treatment of wounds of the heart does not differ from wounds of the chest in general. Absolute rest is essential. It may be advisable to bleed from the arm. If there be a portion of needle or other pointed instrument projecting from the wound in the chest, it should be extracted slowly by alternate movements of traction and rotation so as to favour the clotting of blood in the track made by the instrument. Should there be an accumulation of blood in the pericardium it may be advisable to evacuate it; the use of a trocar and canula for this purpose is condemned because of its incapacity to evacuate blood which has clotted; it does not allow one to identify and secure the bleeding-point, and its use entails the risk of wounding the heart or the pleura. Incision of the pericardium by open operation is the procedure recommended; great care must be taken not to open into one or other of the pleural cavities, especially the left; the soft parts are reflected in the form of a flap having its base at the right border of the sternum; the cartilages of the third, fourth, and fifth left ribs are removed with or without a portion of the sternum. The pericardium is then exposed and may be opened and evacuated; any bleeding-point is seized and ligatured; a wound in the heart-wall may be closed with interrupted sutures; silk is usually preferred because it is more durable than catgut; the sutures should not include the endocardium. Having arrested the hæmorrhage, the wound in the pericardium is sutured, leaving a small opening for drainage, occupied by a strand of gauze. Any pleural complication is thus dealt with before closing the external wound. Should the patient recover, he should be prohibited from attempting any exertion for several months, for fear of the scar yielding and causing aneurysm or rupture of the heart-wall.

Needles have been found in the heart after death, having given rise practically to no symptoms; on other occasions, alarming symptoms have arisen from the passage of a needle through the heart. In one case in which a needle was removed a distinct bruit was audible previous to the operation, and disappeared immediately after.

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1. Acute Cellulitis and Abscess of the Chest-wall.—This may involve the subcutaneous cellular tissue alone, or the deeper layers of tissue beneath the muscles and aponeuroses. The source of infection is not always apparent; the axilla and the upper extremity are regarded as the more common sites of the original infection. The cellulitis may spread over the greater part of one side of the chest, may spread downwards into the abdominal wall or upwards to the shoulder and neck. The pleura and lung may become involved in the infective process.

The disease is to be treated on the same lines as acute cellulitis in other regions, *e.g.* the neck, limbs, pelvis.

Circumscribal acute abscess of the chest-wall is met with in relation to acute osteomyelitis of the ribs, especially that form which follows upon typhoid fever.

2. Cold Abscess of the Chest-Wall.—The common cold abscess of the chest-wall originates in tuberculous disease of a rib or costal cartilage or of the pleura. Less frequently they result from tuberculous disease of the sternum or from the extension of a spinal abscess along an intercostal space. In relation to the ribs and costal cartilages, the abscess may develop on their external surface or on their pleural aspect, or there may be an accumulation of pus on both aspects, communicating with each other across the intercostal space. The channel of communication is often very narrow, and may easily escape detection. While commonly met with in childhood and youth, they may, like tuberculous abscesses elsewhere, be met with at any period of life. They are usually situated on the lateral or antero-lateral aspects of the chest-wall. The clinical features are the same as those of tuberculous abscess in other situations. They have been known to exhibit pulsations transmitted from the heart. When left to themselves they usually make their way to the skin surface, and sooner or later rupture and give rise to a discharging sinus or sinuses. The most satisfactory treatment is to lay the abscess cavity freely open; its walls are then dealt with on general principles; any recess or channel communicating with the cavity must be explored; careful search is necessary to discover the existence of an abscess cavity on the pleural aspect of the ribs or costal cartilages; portions of the latter may require to be removed in order to deal with such a cavity when it is discovered; all diseased bone or cartilage is to be removed. The wound is then closed with sutures, or stuffed with gauze.

3. Tertiary syphilis is met with in the sternum in the form of gumma or cold abscess, or of a sinus leading down to carious bone. Its clinical features and treatment are the same as in syphilitic disease of other bones.

4. Tumours of the chest-wall may be divided into those of the soft parts and those growing from the bones.

Tumours of the soft parts include sebaceous cysts, angiomata, lipomata, molluscum fibrosum, sarcoma, etc.

Tumours originating in the bones include chondromata, osteomata, and various forms of sarcomata. The latter may originate in connection with the sternum or with the ribs; they may give rise to tumours of enormous size; they may project upon the pleural aspect and press upon the lung; the parietal pleura is often involved in the new growth. The removal of a malignant tumour of the chest-wall is always a formidable operation, as it usually entails the removal of a portion of the parietal pleura in addition to several ribs, and exposure of the lung. The skin over the tumour should be reflected in the shape of a large flap, so that it may be possible to hermetically close the pleural cavity at the end of the operation. Should the lung have collapsed during its performance, it may be confidently expected that it will re-expand and resume its functions. While the immediate result of even very formidable operations may be remarkably successful, the patient usually succumbs to recrudescence of the sarcoma at a later period.

Secondary cancer of the sternum and of the ribs is common enough in the advanced stages of cancer of the breast; it is not amenable to surgical interference.

CUTANEOUS AFFECTIONS OF THE CHEST-WALL

The skin of the chest-wall is frequently implicated in the different cutaneous affections. It is the chest that the physician first examines for the eruption of scarlet fever. The rose spots of typhoid fever, while appearing first on the abdomen, if numerous, will also be visible on the chest, the other exanthemata showing likewise their characteristic eruption. The macular syphilide appears upon the chest as an isolated blotch varying from the size of a linseed to about the size of the finger nail, the colour ranging from pink to bluish red or even a brownish tint, not disappearing entirely on pressure. Scattered throughout the blotches or following upon them the papular syphilide may be seen somewhat elevated above the surface, varying in size up to a pea, and of a reddish dusky colour. Herpes zoster, the vesicular eruption found on the chest along the distribution of an intercostal nerve; pityriasis in its various forms; chloasma, and other skin affections, will be found fully discussed under their different sections.

PAIN IN THE CHEST

The descriptive localisation of pain given by different sufferers is frequently very misleading; it is well, therefore, in every case to ask the patient to place the hand on the part implicated. Pain complained of in the chest may have an origin outside the thorax and its contents, as in *Cardialgia* from chronic gastritis or hyperchloridia, the pain resulting from irritation of sensory fibres in the cardiac end of the stomach and the lower end of the œsophagus, either by the products of fermentation and putrefaction (acetic acid, fatty acid, lactic acid) or by the hypersecretion of hydrochloric acid. The pain is then localised at about the junction of the seventh costal cartilage with the sternum, and in the back in the left interscapular region. The pain is of a burning character and is frequently relieved by vomiting. In *hysteria*, especially where there is ovarian irritation, mammary and infra-mammary pain and tenderness is common. The patient shrinks from the slightest touch, especially if her attention be directed to the part; frequently, but by no means always, if the attention be diverted, the part can be handled without producing pain or shrinking. In these cases the mammary tenderness will be accompanied by pain on pressure over the ovaries, increasing at the menstrual period, and frequently with hyperæsthetic areas in the spinal region. Pain in the chest may be of muscular origin. *Myalgia*, the so-called muscular rheumatism, when attacking the intercostal muscles and fasciæ is usually spoken of as *Pleurodynia*. Its connection with rheumatism is not always clear; it may result from strain, fatigue, cold, or other injury to the muscle, and from constitutional causes. The pain may be intense; breathing deeply, coughing, or manipulation of the muscle causing marked increase of the suffering. It is usually localised to one group of muscles. There may be slight pyrexia, but the constitutional symptoms are not marked. A good example of the affection is met after severe coughing, when myalgia may develop in the lower intercostal spaces. The pain of myalgia is frequently relieved by resting the affected muscle, a fact which the sufferer soon discovers, assuming an attitude which will throw the affected group of muscles as far as possible out of action. Pain in the chest may be the result of implication of the intercostal nerves. *Intercostal neuralgia* gives a pain more or less continuous with acute exacerbations, following the line of the intercostal nerve, with tender points near the spine, the mid-axillary line, and near the middle line in front. The pain may be increased by movement, deep breathing, coughing, etc., but is not, as a rule, so much affected by these as in other painful chest affections. Occasionally *intercostal neuritis* may develop; there is more or less severe pain along the line of the affected nerve markedly aggravated by pressure, with possibly other signs of neuritis, and frequently the further development of Herpes zoster. A symmetrical pain along the course of a group of intercostal nerves may develop in the course of affections of the spinal cord and meninges where there is implication of the sensory roots, the most typical example of the condition being found in the so-called girdle pains of tabes dorsalis.

While pain in the chest may result from affections of the chest-wall it is frequently a manifestation of grave disorder of the thoracic organs. The heart and pericardium are not normally endowed with any great degree of sensibility, but in disease the sensibility may be greatly exaggerated. In *Pericarditis* the subjective sensory disturbances vary much. There may be merely a feeling of discomfort and uneasiness in the precordial region, or the pain may be a marked symptom. The pain is usually in the precordial region, but may be localised in the epigastrium. In addition to pain there is hyperæsthesia (see "Pericarditis").

In the affections of the heart, *pain* is an extremely variable quantity. It may be an aching, the protest of an overworked muscle, the intense discomfort and distress resulting from dyspnœa, or the fully developed attack of angina pectoris. The difference is one of degree not of kind. The special diagnostic features will be described in the article "Heart."

Pain of *pleural* origin has also to be differentiated. Here the manner of onset, site, and character of the pain, with the physical signs, readily suffice to distinguish it. Apart from the pain of acute pleurisy, there may be pain of a dull aching character, and persisting for a lengthened period, due to pleural thickening and adhesions, the result of previous inflammation.

Pain from *aneurysm of the aorta* may result from pressure, the tumour implicating a sensory nerve. Much of the pain, however, may be of the nature of a *referred sensation.* The pain from direct pressure will vary with the position of the aneurysmal tumour and the structures implicated.

In *aortitis* pain may be present in the first, second, and third right intercostal spaces. In *mediastinal growths* pain may be severe, and is due as in aneurysm to pressure upon sensory nerves.

Pain in affections of the *lung*, such as pneumonia, phthisis, etc., results, as a rule, from implication of the pleura; a referred pain may, however, be noticed along the course of the intercostal nerves. Pain in inflammatory affections of the chest-wall, mamma, etc., belongs to the domain of surgery.

The diagnosis of *intrathoracic pain* depends upon the diagnosis of the condition from which it results; and to this end the condition of pleura, cardiac muscle, aorta, and pericardium, must be each in turn carefully investigated (see "Pericarditis").

DISORDERS OF THE CIRCULATION IN THE CHEST-WALL

Considerable distension of the veins may be noticed in cases where tricuspid regurgitation is present. In obstruction to the portal circulation as in cirrhosis of the liver, there is a marked distension of the superficial veins in the lower thoracic region along with distension of the superficial abdominal veins; the anastomotic branches of the inferior mammary and epigastric veins relieving the portal system in part, become then enlarged and visible. An enormous enlargement of the veins of the thoracic wall forming an intricate network of distended vessels may be visible when there is obstruction to the return of blood from the chest-wall to the heart. This interference with the circulation may result from the pressure of a tumour (new growth or enlarged glands) on one or all of the great venous trunks or from thrombosis. If the superior vena cava be implicated, the venous distension will be bilateral; if one of the innominate subclavian or axillary trunks, the distension will be unilateral. It must not be forgotten, however, that in nursing women there is usually a considerable dilatation of the superficial thoracic veins during the period of lactation; the same may be seen during pregnancy and menstruation.

Œdema of the Chest-Wall.—Œdema of the chest-wall may be present as part of a general œdema in heart and kidney disorders. There is then considerable œdema of legs, thighs, back, and passing up the posterior aspect of the chest-wall, usually symmetrically, but possibly more marked on one or other side, if the patient has been lying in a lateral position. Œdema localised to the chest-wall, arm, and head may be present when there is obstruction to the venous return, as in mediastinal tumour. A localised œdema of some diagnostic significance may occur in the chest-wall in purulent exudation into the pleura. Thus the writer has seen a localised œdema over the tenth and eleventh rib over an encysted empyema, the œdema being of considerable diagnostic value in the absence of the usual signs of effusion into the pleura. Angio-neurotic œdema is rare in the chest, but may occur. Œdema of the chest is not to be mistaken for myxœdematous swelling of the skin and subcutaneous tissues with their characteristic dry, harsh, and pale appearance, swollen, thickened, and brawny, and not pitting on pressure.

EMPHYSEMA OF THE CHEST-WALL

Subcutaneous emphysema of the chest-wall, a somewhat uncommon occurrence, may result from the passage of air into the tissues, or from the development of gas within the tissues. The latter is the result of bacterial action, and does not here concern us. The passage of air into the tissues may occur under various conditions. In the lung of the child interlobular emphysema is a possible and by no means uncommon accident as the result of whooping-cough or capillary bronchitis, where the cough is frequent, violent, and paroxysmal. When the condition is marked, the air in the interlobular connective tissue may pass along the connective tissue surrounding the bronchi to the root of the lung, and thence diffuse into the mediastinum, neck, trunk, and the general surface of the body. In the adult, the lobules of the lung not being separated by distinct intervals of connective tissue, such an accident is impossible. In the adult the air may enter the mediastinum and subcutaneous tissues through a lesion of some portion of the respiratory or alimentary tract. Ulceration of the larynx, trachea, bronchi or wound of the pleura and lung may result in emphysema of the chest-wall. Given the formation of a cavity in the lung, the surfaces of the pleura having become adherent, the ulcerative process may pass through the parietal pleura, and a sudden effort of coughing produce emphysema of the chest-wall. Thus the air may pass directly from the respiratory tract into the chest-wall or by way of the mediastinum. From the alimentary canal the air may pass into the mediastinum through ulceration of the œsophagus by a malignant growth, or into the abdominal and chest-wall from the stomach or intestine, the viscera becoming adherent to the abdominal wall and the ulcerative process subsequently invading the connective tissue.

In emphysema the skin is pale and elevated above its surroundings. On palpation the part is found to be very soft and yielding, quite unlike the brawny feeling of œdema. There is no pitting on pressure, the indentation made at once disappearing, and on handling the part there is palpable and at times audible crackling. When the air has passed by way of the mediastinum the swelling appears first at the root of the neck, passing up the neck and down over the chest-wall, and so advancing, obliterating the normal outline. When the air under the skin remains aseptic, absorption may take place; if, however, decomposition occurs, very grave complications result. In any case the accident is a very serious one, and, giving rise to distressing dyspnœa, is frequently the final development in an otherwise serious condition.

Tumours of the Chest-wall

These belong mainly to the domain of surgery. There are, however, certain points that must be borne in mind and carefully investigated in cases of obvious localised swellings or new growths. These may be summarised as follows:—

(i.) A careful examination of the condition of the aorta, anteriorly and posteriorly, with the view of eliminating aneurysm.

(ii.) The question of a mediastinal new growth should be considered.

(iii.) If the tumour be a new growth, is it primary or secondary to disease of the viscera, *e.g.* pleura, liver, etc.

(iv.) If there are obvious indications of the presence of fluid, is the condition a superficial localised one, or has it connection with a serous cavity.

Affections of the Muscles of Chest

Myositis, or inflammation of muscle, is rare as a primary disease, but is more common as the secondary result of a septic process; thus the inflammation may go on to suppuration. *Rheumatic myositis* or myalgia is common in the intercostal muscles and fascia. Its differential diagnosis will be found discussed under "Chest Pain." *Trichinosis* is an uncommon disease in Great Britain, but is more common in Germany, and affects the muscles of the chest-wall along with the other muscles of the body. The affection is due to the presence of an embryo parasite in the muscles (see "Trichina Spinalis"). Along with general disturbance and alimentary symptoms there is intense muscular pain, swelling, tenderness, and œdema. If the respiratory muscles become involved, there is dyspnœa. In atrophic conditions of the muscles of the chest-wall fibrillary contractions may be visible, slight momentary contractions, visible, painless, but perceptible to the patient, and best brought out by pressing the muscle or drawing the finger across the surface. Atrophy of the muscles may arise from various causes. It may be part of a general muscular atrophy in a wasting disease, as in phthisis; it may result from separation of the muscle from its trophic centre, as in neuritis; or from destruction of the trophic centre in the cord, as in poliomyelitis anterior acuta. The muscles around the shoulder-joint may atrophy in disease of the joint, the atrophy being usually described as reflex. Progressive muscular atrophies do not as a rule affect the muscles of the thorax.

Paralytic affections of the muscles of the chest-wall are usually the result of lesions of the central nervous system. Partial paralysis of the intercostal muscles may be noticed in hemiplegia, but the fully developed bilateral paralysis occurs in its typical form in transverse myelitis of the cervical region of the cord. There is then entire paralysis of the intercostals with loss of movement in the chest, the respiratory movements being entirely abdominal. In diphtheritic paralysis the intercostal muscles and diaphragm may be involved, rendering respiration difficult or impossible. Local paralysis of individual muscles may occur, for example the pectoralis major, from implication of the nerve trunk to the muscle. Hypertrophy of the muscles of the chest may, up to a certain point, result from overuse, as is seen in the respiratory muscles in emphysema.

Cheyne-Stokes' Respiration. *See* RESPIRATION.

Chicken-Pox. *See* VARICELLA.

Chilblain. *See* ERYTHEMA.

Children, The Development and Clinical Examination of.

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THE child differs from the adult not only in his small size, his softness, and his inability to do things, but also, very importantly, in the fact that, if he is healthy, he is constantly growing in bulk, in endurance, and in all sorts of capacity. In examining sick children, therefore, we have not only to face the ordinary problems of clinical medicine, but we are also continually being met with the question, Is the patient normal for his age as regards growth and development? If we cannot satisfy ourselves on this point, we are likely to miss much that is of importance in the case. In dealing with the investigation of children, therefore, we shall begin with a short account of such facts of growth and development as have important clinical bearings, and then proceed to consider the examination of the various organs and systems.

GROWTH IN WEIGHT.—At birth the baby weighs, on an average, about 7 lbs. (5 to 12). During the first two days there is a loss of from 8 to 10 oz., resulting partly from the passage of urine and meconium, and partly from the fact that the child does not receive enough nourishment at first to make up for the tissue waste. On the third day a gradual increase begins, the birth-weight being reached again about the tenth day of life, and after this the rise continues more or less steadily. The gain may vary considerably on different days, but the average daily increase is about ⅔–1 oz. during the first five months, and from ⅓–⅔ oz. during the rest of the first year.

By the end of the fourth month the baby's weight should be nearly double, and by the end of the first year about three times its original figure. During the second year the child gains from 5 to 6 lbs.; during the third, about 4½ lbs., and during the fourth, fifth, and sixth, about 4 lbs. a year. Thus by the end of the sixth year the weight is about six times, and at fourteen years twelve times, as great as at birth.

The advantage, from a clinical point of view, of weighing infants regularly is very great. By it we can gauge more simply and surely than in any other way the extent to which a particular diet is being assimilated. If a young child is losing weight, or even not gaining it for some weeks, this is to be regarded as an important morbid symptom, and its cause searched for.

GROWTH IN LENGTH.—The average length of a new-born baby is 19½ or 20 in. During the first six months he grows from 4 to 5 in., and in the second, 3 to 4 in. During the second year he gains from 3 to 5 in.; during the third, 2 to 3½; and during the fourth, 2 to 3. After this the gain is rather less, and amounts to 1⅔ to 2 in. every year. By the end of the fifth year the child has generally doubled his original length.

Severe chronic dyspeptic disturbance and any other morbid conditions which profoundly interfere with the general health are apt to lead to dwarfing. Rickets, if severe during early infancy, has often this effect. Chronic disease or defect of the brain generally interferes markedly with the growth of the body.

DENTITION.—(*A*) *The Temporary Teeth.*—The temporary or milk teeth are twenty in number. The following may be given as the usual ages at which they appear:—

(1) Lower central incisors	6 to 9 months.
(2) Upper central and upper lateral incisors	8 to 11 „
(3) Lower lateral incisors, and lower and upper first molars	12 to 15 „
(4) Lower and upper canines	18 to 24 „
(5) Lower and upper second molars	24 to 30 „

The teeth normally come in pairs—a tooth on one side cutting the gum about the same time as the corresponding one on the other side. Those in the lower jaw appear a little earlier than the corresponding ones above, except in the case of the lateral incisors. The intervals between the cutting of the different groups of teeth may vary considerably in healthy children, but the order in which they appear seldom varies. Irregularity in their order, and the appearance of teeth singly, instead of in pairs, is an indication of rickets. Sometimes teething begins unusually early, and infants may even be born with one or more teeth through the gum. Early dentition has no special clinical significance. Delayed dentition sometimes occurs without any apparent interference with the child's health. Generally, however, it is attributable to rickets, and this disease should always be suspected if a child has no teeth at ten months.

Symptoms of Teething.—In many cases nothing unusual is noticed in the general condition of the child while the teeth are appearing through the gum, and there is little or no local disturbance. Often, however, there are signs that the baby has pains in its jaw when the teeth are coming, and there may also be localised inflammation of the gum, and greatly increased secretion of saliva. With or without these local symptoms we often find rise of temperature, restlessness, loss of sleep, and irritability. The appetite also may be lost, and there may be constipation or slight diarrhœa. Often the child loses weight. Less frequently slight temporary neuroses develop, *e.g.* there may be a constant winking of the eyes, or a frequently recurring cough or rapidity of breathing without any pulmonary disease.

Some children show a tendency to certain diseases at the time of teething which they do not seem to have at other times. Thus it is not very uncommon to find a child who with the appearance of each new set of teeth has an attack of diarrhœa or bronchitis which resists treatment stubbornly while the teeth are in process of appearing, but which rapidly recovers (under otherwise similar conditions) when they are through. Similarly, we see infants with eczema who have a marked relapse with each new group of teeth; and often an obstinate eruption will disappear almost spontaneously whenever all the teeth have pierced the gum.

The place of dentition as a factor in the causation of disease is a matter which has been much disputed. There can be no doubt that teething is not in itself a cause of death, and that its influence in producing and predisposing to disease has been enormously exaggerated. At the same time it seems equally certain that teething, like menstruation, pregnancy, and other natural states, is often accompanied by marked symptoms both local and reflex, and like them may produce temporarily a tendency to disease which is not present at other times. When symptoms due to teething cause alarm, this is not because they are themselves such as to threaten life, but because they are apt to lead us to suspect the presence of some serious disease. Thus an attack of acute bronchitis is probably in no respect more serious in a teething infant than in one who is not teething, but in the former there may be an increase in the rapidity of the breathing and an amount of fever present which make the case simulate one of pneumonia.

The diagnosis that the symptoms in a case are due to dentition can rarely be made with any confidence until the patient is well on the way to recovery

Treatment.—Lancing the gums was at one time very largely practised. Now it is not considered advisable, except occasionally when the gum is swollen, red, and tense over a coming tooth, and the child is suffering local pain or showing signs of reflex nervous disturbance. Under these circumstances it sometimes gives marked relief and can do no harm.

The general restlessness and irritability of teething children may be relieved by a few grains of antipyrin, phenacetin, or bromide. An aperient generally relieves the symptoms greatly.

(*B*) *The Permanent Teeth.*—The permanent teeth number thirty-two. The order and usual time of their appearance are as follows:—

First molars	6 years.
Incisors	7 to 8 „
Bicuspids	9 to 10 „
Canines	11 to 13 „
Second molars . . .	12 to 15 „
Third molars (wisdom teeth) .	17 to 25 „

The eruption of the permanent teeth is not a usual source of irritation either local or general in childhood; but the wisdom teeth, especially those of the lower jaw, may cause some distress when they appear.

DEVELOPMENT OF VARIOUS GLANDULAR ORGANS.—*The Salivary Glands and the Pancreas.*—Not only is the saliva very scanty in young babies, but it is also deficient in diastatic power. After the third or fourth month its amount and its functional capacity increase, but it is only towards the end of the first year, when a number of teeth have usually appeared, that its amylolytic action becomes at all fully established. The action of the pancreatic secretion on starch is practically absent at birth, and develops, *pari passu*, with that of the saliva. The pancreatic juice is relatively active in digesting proteids and fats even in new-born infants.

The Stomach.—At birth the stomach has its fundus only slightly developed, so that it has a tubular shape and a very small capacity. According to Holt, the average infant's stomach can contain at birth only 1⅛ oz., at three months 4½ oz., at six months 6 oz., and at twelve months 9 oz.

The stomach plays a less important part in the process of digestion during infancy than in later life. This is owing to the gastric juice being scantier and less powerful, and to the fact that the stomach contents are not allowed to remain very long in it. The relatively small proportion of hydrochloric acid in the gastric juice of infants probably helps to account for their characteristic susceptibility to gastro-intestinal infection.

The Intestine.—In infants the intestine is relatively long, and its muscular wall comparatively feebly developed. This latter fact helps to explain the greater tendency to constipation and to flatulent distension in young children.

The Thymus.—The thymus gland is a large organ at birth, weighing about half an ounce, and it grows until, by the end of the second year, it weighs 1½ to 2 oz.; after that age it gradually diminishes. The area of dulness over the manubrium sterni which it causes must not be forgotten in examining the thorax in babies.

DEVELOPMENT OF THE SPECIAL SENSES.—*Eyesight.*—Within a few weeks of birth most infants are evidently pleased by a brightly coloured object or a light. By the third month the child should show that he recognises his mother's face, and after that he soon gets to know the look of many things. He is long, however, of being able to distinguish colours. He may know red and yellow in the first twelve months, but will not probably recognise blue and green until the second or third year.

In testing the sight in an infant, we may try if he follows a lighted match or other bright object with his eyes, whether he seems to recognise his mother and to see familiar objects, such as toys or food, approaching. It should also be noticed if his pupils contract with light and, if he is more than a few months old, with accommodation. If the point of a finger is suddenly brought close to the eye of a baby over two months old who has normal sight, it causes winking.

Hearing.—During the first day or two of life all children are deaf; but by the second week they should be able to hear loud noises quite well. If they show no signs of doing so, it may be suspected that they are either deaf or idiotic. Although children are very early attracted and pleased by noises, they cannot usually distinguish even their own mother's voice when three months old.

Taste and smell are both relatively well developed within the first few days of life. If they have to be investigated in childhood, familiar articles of food generally form the best tests.

Sensibility to touch, temperature, and pain are not very acute in early infancy. To be satisfactory, the examination of the various forms of sensation in infancy must be not only carefully made but frequently repeated.

DEVELOPMENT OF THE VOLUNTARY MOTOR FUNCTIONS.—At birth the infant's actions are automatic, and it is only when some three or four months have passed that he acquires the power to execute distinctly voluntary movements, such as turning towards those he is fond of, or pushing away from him something he does not like. By noticing the age at which a child acquires the power to execute certain movements, we gain information as to his muscular, and to some extent also as to his mental development.

If a finger is laid in an infant's palm it is generally tightly grasped, and if the child is over five months old it will probably also be carried towards his mouth. Should the child's fingers show no inclination to close on an object placed in his palm, it is a morbid sign suggesting usually either paralysis or great mental impairment.

A healthy, normally developed baby ought to be able to hold his head up when he is three or four months old, according to the degree of his muscular vigour. Only when he is eleven or twelve months old does he permanently acquire the capacity for sitting unsupported. Some children creep before they walk, as early perhaps as the ninth month, others much later. A strong baby generally begins to try to stand by the ninth or tenth month, and he may be able to do so by himself by the eleventh or twelfth. Some children can walk before the end of the first year, others not till they are nearly eighteen months old; fourteen or fifteen months is perhaps the average age.

Any delay in the acquisition of these ordinary muscular actions requires investigation. It may be accounted for by the weakening influence of a recent illness, or by some lesion of the bones, joints, or muscles. More commonly, however, it is found to indicate the presence either of rickets or of mental deficiency.

DEVELOPMENT OF SPEECH.—When the infant is twelve months old he will understand a good many words, and may be able himself to use one or two with a definite meaning. During the second year his knowledge of words increases fast, and before the end of it he may have begun to use short phrases. The age, however, at which normal children learn to speak varies very greatly.

Should a child be unable to speak by the time he is three years old, the cause of this should be investigated. If he has suffered much from illness, the backwardness in speaking may be due to this only, and will in that case pass off as he regains strength. The hearing should be carefully examined; comparatively slight deafness may interfere a great deal with a child's progress in learning to speak. Perhaps the commonest cause of delayed speech is intellectual deficiency; the mental condition should, therefore, always be examined in such cases (see "Aphasia," "Deafmutism").

Order and Method of Clinical Examination.—When our patients are little children we have, while using the ordinary methods, to shorten our examination as far as possible so as not to exhaust them, and also to avoid very carefully doing anything to cause them fright or annoyance. As a general rule, while in examining adults we proceed *system by system*, investigating in turn the alimentary, circulatory, respiratory, and other organs, in young children we go rather *by methods.* We inspect first as much as we can without touching, then we palpate all over, then auscultate, and so on. There are, however, of course, many exceptions to this rule.

Before commencing the physical examination it is always well to make a few *preliminary inquiries.* It is important, for example, first to ascertain clearly for which of the child's ailments medical advice is sought, and how long the illness has lasted. The family history is also to be inquired into, the health of the parents and other children, the state of the mother during her pregnancy, and the nature of the labour when the child was born. The previous health, development, and feeding from birth onwards are very important, and special inquiries must be made as to any symptom of congenital syphilis, and as to the dates of occurrence of any of the infectious diseases. Full details of the nature and order of onset of the symptoms of the present illness should always, if possible, be obtained.

While these inquiries are being made, the child (who should, if possible, be seated on his mother's or nurse's knee) has time to grow accustomed to the doctor's presence. At the same time the medical man, without coming too near, may learn much to guide him in his further examination of the child. Physiognomical diagnosis plays a far greater part in infancy than it does in later life, and the physician who is practised in this art will always be at an advantage. He must not, however, trust to it for such information as can only be acquired with accuracy from the ordinary and more laborious methods of examination. The state of development and nutrition, the complexion and expression, and the form of the head and other uncovered parts are to be specially noticed, as well as any traces of rickets, syphilis, or other disease. The child's demeanour and the attitude he assumes are also very significant. The number and character of the respirations should be observed, and the nature of the cough and of the voice in speaking or crying is also worthy of notice.

Then comes *palpation*, and the pulse is generally felt first, lest its rate should be altered by fright or annoyance due to the further examination. A shy child's pulse will be easiest felt while his mother keeps his hand in hers. The abdomen and thorax are next palpated, without the child's position being changed (at first, at any rate). The hand is also passed over the chest to feel the amount of rickety beading, if any is present, and the position and character of the heart's impulse. The consistence of the muscles and the mobility of the joints should next be examined, and the state of the ossification of the cranium and possible presence of enlarged glands in the neck should not be overlooked.

Auscultation should generally be practised before percussion, as being less likely to cause annoyance, and immediate auscultation is sometimes less resented than the use of a stethoscope.

Then comes *percussion;* and lastly the mouth and tongue have to be inspected, and the gums and fauces seen, and, if necessary, felt. This is the part of the examination which is most likely to make the child cry, and therefore it is left to the end. The *temperature* may be taken at any time before or during the examination.

THE HEAD.—In examining the head we must note its size, shape, and ossification, including the condition of the fontanelle and sutures.

Size.—At birth the average circumference of the infant's head is 13 to 13¾ inches. During the first six months it gains about 3 inches. At the end of the first year the head measures about 18 inches; at two years old about 19; at five 20 to 20½; and at ten years about 21 inches. There are, however, considerable variations in the size of the normal child's head. As a general rule, where the head is unusually large or small from causes which interfere with the health of the brain it has also a characteristic shape.

Shape.—In rickets the cranium is square and sometimes asymmetrical; sometimes it presents a natiform or bossed appearance (see "Rickets"). The hydrocephalic cranium is large and rounded; that of microcephalus small, with a receding forehead and a pointed vertex.

Ossification.—This is investigated by palpating the anterior fontanelle, the sutures, and the back of the head (for craniotabes).

The Fontanelle.—The *normal fontanelle* (*i.e.* anterior fontanelle) is rhomboid in shape with not very thin edges, and its membrane is stretched somewhat tensely between these, so that its surface is about the level of the surrounding bones. It presents a slight pulsation transmitted to it from the arteries at the base of the brain, and a systolic murmur is often audible over it. As the child grows older the fontanelle gradually diminishes in size, and it is usually closed between the fifteenth and eighteenth month, or at latest before the end of the second year. It is changed in various ways in disease. If there is much *thinning of the bony edges* of the fontanelle, this indicates rickets or long-continued increase of the intracranial pressure.

Alterations in the tension and level of the membrane give valuable information as to the state of the cranial contents. Slight increase of tension, with bulging, indicates cerebral hyperæmia, active or passive, and is therefore met with in fever, in whooping-cough and bronchitis, and always temporarily when the child coughs or cries. Great tension with marked bulging means considerable increase in the contents of the skull, and is found with hydrocephalus, intracranial tumour, and cerebral hæmorrhage. Abnormal depression of the membrane indicates lowering of the vital powers. It is met with in acute diarrhœa and in other exhausting conditions, which call for stimulant and supporting treatment. When an infant presents symptoms which lead one to suspect intracranial disease, a normal condition of the fontanelle is always a reassuring sign.

Alterations in the size and date of closing of the fontanelle are also important. Great delay in closure, so that the fontanelle is too large for the age of the child, is met with in rickets, in chronic hydrocephalus, and in cretinism. Premature closure is a valuable early sign of microcephalus.

The Sutures.—Any gaping of the sutures or thinning of their bony margins has the same significance as enlargement of the fontanelle.

THE NECK.—The state of the lymphatic glands in the neck should be noted as a matter of routine. If any are enlarged, the area of skin or mucous membrane connected with these must be carefully examined.

Stiffness of the neck must not be overlooked. Sometimes it is due to muscular rheumatism, sometimes to cervical caries. It is sometimes a symptom of basal meningitis, or some other intracranial disease.

THE BACK.—In addition to the characteristic curvatures due to Pott's disease and rickets, we have to look out for the apparent kyphosis which is due to paralysis of the back muscles from any cause. We have also to remember that inability to hold the back straight is a common symptom of idiocy. Any lack of suppleness and any tenderness on free movement of the spine should arouse suspicion of tuberculous spine-disease.

THE LIMBS.—The state of the circulation in the extremities, the development of the muscles, the configuration of the bones, and the size and movements of the joints have all to be examined. Pain on movement of one or more limbs may be an early and important sign of infantile scurvy, or of syphilitic epiphysitis, or may be due to a subperiosteal fracture. Clubbing of the finger-ends may throw considerable light on the nature of an obscure heart or lung case, and the presence of tuberculous or syphilitic disease of a finger may help to elucidate the nature of a brain lesion. In children with any manifestation of rheumatism, careful search should always be made over the bony prominences of the limbs for rheumatic nodules.

THE MOUTH AND THROAT.—*The Lips.*—The appearance of the lips is mainly useful as an index of the state of the circulatory system (anæmia, cyanosis, etc.)

The Tongue.—In young infants the tongue is more or less coated, owing to the scantiness of the saliva. In older children we often meet with the so-called "mapped" or "geographical tongue." This condition has nothing to do with syphilis, and has, indeed, little practical importance.

The Teeth.—In examining the teeth we have to note the stage of progress of dentition and also any abnormalities of form such as those characteristic of congenital syphilis. Flattening of the tips of the more prominent teeth in both jaws indicates that the patient grinds his teeth.

The Gums.—Various forms of stomatitis (*q.v.*) exert their most characteristic effects on the gums. Sponginess of the gums should always suggest the probability of scurvy being present, although sometimes it occurs as a merely local condition.

The Palate.—In examining the hard palate in very young infants we often see little yellowish nodules in the mucous membrane near the mesial line. These are called "epithelial pearls," being composed of degenerated epithelial cells. They have no clinical significance, and rapidly disappear.

The Throat.—In feverish cases of any kind the examination of the throat must never be omitted. In carrying this out it is of great importance to ensure, to begin with, that the child is in such a position that, when his mouth is opened, the light will at once fall on the back of the throat. It is also necessary to take precautions against possible struggling on the part of the patient. The handle of an ordinary spoon forms the best tongue-depressor, being less likely to alarm the child than any special spatula.

Digital examination of the pharynx, fauces, and naso-pharynx is very important, especially in young infants, in whom it is often difficult to get a satisfactory view of these parts. It is especially called for when there is a possibility of retropharyngeal abscess.

THE ABDOMEN.—*Inspection.*—In young children the abdomen is normally more prominent than in the adult. This is due partly to the relatively large size of the liver and the narrowness of the thorax, and partly to the fact that the bowels are more readily distended by flatulence owing to the weakness of their walls and those of the abdomen. Any dilatation of the superficial veins and any redness about the umbilical region must be noticed.

Retraction or hollowing out of the abdomen is a very significant sign of cerebral disease.

Palpation.—Tenderness on palpation of the abdomen, if at all well marked, usually indicates the presence of peritonitis or some other form of inflammation. Enlargement of the abdominal organs and tumours are generally easily felt, provided the children do not resist, and a combined rectal and abdominal examination is especially useful. In all cases of difficulty it is advisable to give chloroform.

Percussion.—Percussion is useful in determining the state of the stomach and bowels and in confirming the results of palpation. It is also of great value in ascertaining the presence of free fluid in the peritoneum.

The Liver.—The lower margin of the liver can usually be made out by palpation as well as percussion. It reaches a little below the costal margin in the right mamillary line. Diminution in size of the liver is very rare. Enlargement is common, and may be due to fatty accumulation, waxy disease, cirrhosis, passive congestion, and various other causes.

The Spleen.—The spleen is best investigated in children by palpation. To examine it you stand on the child's right side, and, placing the right hand on the left side of the abdomen with the first two fingers over the left hypochondrium, press gently inwards and upwards. In some cases where the spleen is normal in size, and always when it is enlarged, its rounded edge will be felt as a soft and readily movable body. If the lower edge is distinctly below the level of the ribs the organ may be regarded as abnormally large. Sometimes it extends right down into the pelvis. Enlargement of the spleen is common, and may be due to syphilis, leucæmia, pseudo-leucæmia, typhoid fever, tuberculosis, hepatic cirrhosis, and other causes.

The Mesenteric Glands.—Tuberculosis of the mesenteric glands is an extremely common condition from a pathologist's point of view. Clinically, however, it is only occasionally that we are able to make sure of its presence during life, as in many of those cases in which the glands are most enlarged there are other changes in the abdominal cavity which render them difficult of palpation.

EXAMINATION OF THE FÆCES.—The *meconium* which the infant passes during the first three or four days is of a dark greenish-brown colour, of a viscid semi-solid consistence, slightly acid in reaction, and without odour. It is sterile at birth, but within a few hours micro-organisms find their way into it through the anus. After four or five days the motions cease to contain meconium and assume the characters of normal infantile fæces.

Normal Fæces.—In a healthy breast-fed baby the motions are from two to four in number daily during the first month or two, and usually two, but sometimes only one, daily after that. They are of an orange-yellow colour, and of a uniform semi-solid consistence. Their reaction is acid, and they have a slightly sour but not offensive odour. The stools of a hand-fed infant are similar, so long as his food resembles breast-milk in composition and is well digested.

The motions of a healthy child may vary in number, consistence, colour, reaction, and odour according to the character of the food given him. The number of the motions is increased and the consistence lessened by increase of cream in the food, and they become fewer and more solid if the cream is lessened or the casein increased. The colour depends for its shade largely on the percentage of fat, being lighter if this is small in amount. The darkening of the fæces by bismuth and iron is to be remembered. The reaction may become alkaline from changes taking place in the incompletely digested proteids. The addition of beef-tea or raw-meat juice to the diet of a milk-fed baby causes the fæces to acquire an offensive odour.

By the end of the first dentition the motions have assumed a brownish colour, and are usually formed.

Abnormal Fæces.—The appearance, reaction, and other characters of the motions may be greatly changed by disease (see "Gastro-enteritis"). When a large amount of *mucus* is recognisable by the naked eye in the motions, it generally indicates disease of the large intestine.

Pus is found in the stools in cases of ulceration of the bowel and also in severe catarrh.

Blood is passed under a great variety of conditions. When pure blood comes in any quantity from a child's bowel, apart from diarrhœa or ulceration, it generally comes from a rectal polypus. In young infants the presence of a small amount of blood in the motions is often due to an anal fissure. Copious hæmorrhage from the stomach and bowel in new-born children (melæna neonatorum) is fortunately rare, but it is comparatively common to meet with cases of spurious melæna in which blood which has come from the nose or elsewhere has been swallowed and has been passed with the motions.

Fragments of *membrane* are found in the stools in dysentery and in croupous enteritis. *Worms* and their *ova* are often present, and foreign bodies such as earth, sand, stones, etc., may be found in the case of children who suffer from pica.

THE CIRCULATORY SYSTEM.—*The Pulse.*—The following may be given as the average *pulse-rate* in healthy children at different ages during sleep or perfect quiet:—At birth, 140 to 120 per minute; 6 to 12 months, 115 to 105 per minute; 2 to 6 years, 105 to 90 per minute; 11 to 14 years, 85 to 75 per minute. Mental emotion and bodily exercise may quicken the pulse as much as twenty or thirty beats in the minute; hence the great importance of counting it when the child is undisturbed. While the pulse-rate itself is often of little significance, its ratio to the respiration-rate is always important.

Irregularity of the pulse is not uncommon in children, and is generally of little importance. If, however, the pulse is slow as well as irregular it is worthy of careful attention. Such a condition of pulse sometimes occurs after acute illnesses without any serious significance, but it is also a common early symptom of intracranial disease.

The Heart.—The heart lies more horizontally in infancy than in later life, consequently the apex-beat in children under four years is generally outside the nipple in the fourth intercostal space. The area of the deep, as well as that of the superficial, cardiac dulness is relatively large in childhood. The normal heart-sounds in little children are peculiar in certain ways. The first sound is louder than the second in all the areas. The pulmonary second sound over the base is normally louder than the aortic. It is not to be regarded as accentuated unless it is found to be constantly louder than the first sounds over the base, when the child is at rest. Owing to the greater conductivity of the tissues the heart-sounds are heard more distinctly all over the thorax in young children than in adults.

When heart-murmurs are found they may be functional in character, or due to congenital malformation or to the result of endocarditis. Functional heart-murmurs are very rare in young children; those due to congenital malformations are, of course, much commoner than in later life. When valvular disease is present it is always of importance to ascertain, if possible, whether or not it is of rheumatic origin. For this purpose inquiry must be made as to former rheumatic manifestations and the patient examined for present signs of the disease, especially for rheumatic nodules.

Respiratory System.—*Inspection—Form of the Chest.*—In the normal infant the chest is more cylindrical than in the adult, and its section is consequently more nearly circular in outline. Its shape is readily altered by anything that tends to soften the already soft chest-wall or to interfere with the free expansion of one or both lungs. The commonest alteration which we meet with is the deformity characteristic of rickets, but we also find various degrees of pigeon-breast, and occasionally, as in adults, unilateral retraction or distension, due to pulmonary or pleural disease or to spinal curvature.

Noticeable enlargement of the superficial veins over the upper part of the chest is a common symptom of enlarged bronchial glands.

Movements of the Chest.—(*a*) *Their Character.*—The respiratory movements of the chest are slight in infants, because the type of respiration in them is almost entirely abdominal. Indrawing of the epigastrium and adjacent parts on inspiration indicates, with a normal chest, that sufficient air is not entering the lungs, and is an important sign. In broncho-pneumonia it helps us to gauge the extent to which the lung is affected, while in croup its presence to any marked degree is one of the main indications for immediate surgical assistance.

If the chest is abnormally collapsible from rickets, this symptom may be present to a considerable degree without indicating any serious risk.

(*b*) *Rate of the Respiration.*—In young infants the rate of breathing is very variable, and it is difficult, therefore, to estimate it correctly. At birth the number of respirations per minute varies from 32 to 50, and during the first year from 25 to 35. During the second, third, and fourth years it is about 25 per minute. It varies, like the pulse, with the temperature and with the mental state.

Generally the important point to determine is not merely the rate of the breathing, but the ratio between that and the pulse-rate. The pulse-respiration ratio should in health be 1 to 3½ or 4, and any great disturbance of these proportions is of clinical significance. Increased respiration-rate with dyspnœa signifies pulmonary disease. Rapid breathing without dyspnœa may have the same significance, or it may arise from abdominal distension or rickety deformity of the chest. It is also met with, and sometimes to a marked extent, in the irritable states which sometimes accompany teething and lithæmia.

Extra-auscultation.—Before and during the physical examination of the child's chest it is very important to listen carefully to his breathing, his cry, and his cough.

(1.) *The Breathing.*—*Snuffling breathing* may be due to ordinary catarrh. When, however, it persists for a long period, or occurs apart from other catarrhal symptoms, it always suggests the presence of syphilis.

Snoring during sleep, with noisy breathing while awake, and a nasal tone of voice, commonly indicate the presence of enlarged tonsils or adenoids, but may be due to diphtheritic paralysis of the palate. *Noisy breathing with dyspnœa*, chiefly during inspiration, and a cry which is nasal but not generally hoarse, is characteristic of retro-pharyngeal abscess.

Deep *sighing* may mean very little, but it is one of the symptoms met with in the prodromal stage of tuberculous meningitis.

Laryngeal or stridulous breathing in young infants is most frequently due to congenital laryngeal stridor, but may be caused by laryngitis or enlarged bronchial glands. In older children it often indicates the presence of true or false croup, or of some other form of laryngeal obstruction such as polypi.

Bronchial wheezing is often readily audible in bronchitis of the larger tubes as well as in asthma.

(2.) *The Cry.*—From the loudness of an infant's cry we can gauge his strength to a certain extent. If he cries loud and long we may be almost sure that he has no serious acute condition of his respiratory passages. A hoarse laryngeal cry is generally a symptom of congenital syphilis in young infants.

(3.) *The Cough.*—The cough is *loud and clanging* at the beginning of an attack of croup, and husky and stridulous at a later stage. In bronchitis it is often *deep and harsh.* In pneumonia, with accompanying pleurisy, it is *suppressed and painful.* If a child coughs loudly without wincing, you may be sure that he has not got acute pleurisy. A loud, noisy cough at night and in the morning is characteristic of a catarrhal condition of the throat with or without dyspepsia. If the cough has a distinctly *paroxysmal* character, this is always suggestive of whooping-cough, especially if it is worse at night, and sometimes ends in vomiting. Markedly paroxysmal coughs, however, are also met with in empyema, in enlargement of the bronchial glands, and in some cases of simple bronchitis.

The Sputum.—It is only when children are between five and seven years old that they begin to expectorate naturally. Before that age they swallow the sputum.

Real hæmoptysis is rare in childhood. In phthisis it only occurs as a late symptom. It is most frequently met with in whooping-cough.

PHYSICAL EXAMINATION OF CHEST.—*Palpation.*—In examining the lungs, one of the first things to do is to ascertain the position of the *heart's apex-beat.* If it is displaced, this throws important light on the state of the lungs.

Vocal fremitus is often difficult to obtain in children, owing to the quality of the child's voice and his unwillingness to speak loud. In infants it is often got very satisfactorily during crying.

Auscultation.—Little children often hold their breath when you try to auscultate them. This is annoying, but at least it proves that there is no serious lung-disease present. Loud crying does not interfere with auscultation very much. The long breaths which it necessitates make any accompaniments to the breath-sounds more clearly heard, and the pauses between the cries usually allow time to hear the heart-sounds during one or two cardiac revolutions. Whimpering and sobbing are much more troublesome.

The *breath-sounds* in young babies are naturally very weak, owing to the feeble, shallow nature of their breathing. As the children grow older they get gradually louder, and by about the age of six months they have acquired the peculiar harsh character known as *puerile.*

If on auscultating a child's chest the breath-sounds are found to differ in loudness on the two sides, the side with the weaker breathing is almost always the abnormal one. *Abnormal weakness* of the breathing is met with in the early stages of pneumonia, in pleuritic effusion, in collapse of the lung, and in pneumothorax.

Tubular breathing is more often met with in pleurisy with effusion in children than in adults, and sometimes leads to a mistaken diagnosis of consolidation.

Friction is sometimes difficult to make sure of in young babies, and we have to depend on the catch in the breath and the evident pain accompanying it to confirm our diagnosis. A peculiar *expiratory rhythm* of the breathing is very characteristic of childhood. In this there is a loud, long expiration followed immediately by a short inspiration, then a pause, then the loud expiration again, and so on. The pause occurs, therefore, after inspiration, instead of, as normally happens, after expiration. This type of breathing is often noticed in normal children—especially when they are frightened. It is seen markedly in pneumonia, and in it the expirations have frequently a grunting character.

Vocal resonance can generally be obtained best in young children when they are crying.

Percussion.—In children a light stroke should always be used in percussion. This is advisable, not only because it is less apt to annoy the child, but also because it is more likely to lead to accurate results. Strong percussion over a part often misleads by bringing out dulness or resonance from underlying organs.

It is essential to see that the patient is *sitting straight.* Even a slight twist of the spine may give rise to distinct differences of percussion-note on the two sides of the chest. We must also beware of the differences due to abnormal curves of the ribs. Too much importance must not be attached to small areas of partial dulness in a child's chest; but at the same time it is to be remembered that the presence of fluid in the pleura often causes less absolute dulness in children than might be expected.

A well-marked *cracked-pot sound* can often be obtained in little children who have perfectly normal lungs—especially when they are crying.

URINARY SYSTEM.—In young children the bladder is higher than in later life. This must always be remembered in tapping the abdomen.

Micturition.—The infant may pass water very soon after birth, but often does not do so for twelve or even twenty-four hours. During early infancy incontinence is the normal condition. Some infants have acquired a certain control over their bladder by eighteen months old; in others this comes considerably later. If, however, a child cannot retain his urine to a considerable extent, during his waking hours, by the time he has reached his third year, he may be said to suffer from incontinence.

Incontinence of urine may be due in girls to a local malformation, and in both sexes to conditions of general debility or to various severe organic diseases of the nervous system. In the great majority of cases, however, it is a functional neurosis (see "Urine").

Retention of urine is much less common. It may be the result of extreme phimosis or of an impacted calculus. It may also be due to reflex irritation from an anal fissure, or from threadworms, or to central causes, such as meningitis.

Dysuria is not uncommonly due in infants, as well as in older children, to acid urine. In boys it is sometimes caused by phimosis or preputial adhesions. *Renal colic* from the passage of uric-acid crystals is occasionally met with.

The Urine.—In infants some idea of the colour and amount of the urine passed may be obtained by examining the napkins; but, where it is possible, a proper specimen should be collected.

The following table (Holt) gives the average amounts passed at different ages:—

	Ounces.
First twenty-four hours	0 to 2
Second twenty-four hours	⅓ „ 3
Three to six days	3 „ 8
Seven days to two months	5 „ 13
Two to six months	7 „ 16
Six months to two years	8 „ 20
Two to five years	16 „ 26
Five to eight years	20 „ 40
Eight to fourteen years	32 „ 48

Albuminuria is sometimes present normally during the first ten days, owing, apparently, to the passage of uric-acid crystals. In older children its significance is the same as in adults. All cases of albuminuria should be examined for traces of recent scarlatina.

Hœmaturia is found in acute nephritis, diseases of the bladder, and tumour of the kidney. It is also met with in infantile scurvy, purpura, malaria, and various other conditions.

Pyuria occurs in cystitis and pyelitis, in both cases usually with acid urine.

Glycosuria is rarely found to any great degree in childhood.

THE INTEGUMENTARY SYSTEM.—The colour of the skin as well as its state as to moisture and temperature must always be noted; and it must be examined for œdema, desquamation, and eruptions.

Cyanosis setting in during an acute illness is often of great importance as indicating failure of the heart or the degree to which the lungs are affected. It is also a characteristic symptom of various forms of blood-poisoning. When habitually present it generally indicates the presence of a congenital heart-lesion. The peculiar sallow tint of the skin in infantile scurvy, congenital syphilis, and other cachectic conditions, is often of great assistance in diagnosis.

Undue perspiration in young children is usually a sign of rickets, although it also occurs in various forms of debility and in blood-poisoning. A cold, clammy state of the extremities should draw attention to the state of the digestion and general health.

THE TEMPERATURE.—In young children it is generally more convenient to take the temperature in the groin than in the axilla, because of the attitude they assume in sitting and the disposition of their clothes. In important cases the rectum should be preferred. The thermometer should never be trusted in the mouth in young children.

The *normal temperature* shows greater daily variations in infancy than in adult life, but its average level is about the same. There is a greater tendency for the temperature to vary upwards and downwards on slight occasion.

A *subnormal temperature* is characteristic of atrophy. In wasted babies, therefore, a temperature of 98° or 99° F. may denote the presence of fever.

Pyrexia is sometimes caused by emotional excitement, and sudden rises of temperature are often due to trivial causes. Continuous pyrexia, however, is always important. A high temperature occurring between the second and fifth day after birth, unaccompanied by other obvious symptoms of illness, is a sign that the infant is not getting sufficient nourishment.

THE NERVOUS SYSTEM.—In investigating the presence of nervous disease in children we have not only to examine for the usual physical signs, but also, especially in acute cases, to make careful inquiry for a history of various suggestive symptoms.

Thus we may ask if any change has been noticed recently in the child's disposition and temper, whether he has complained of pain in the head or elsewhere, or has had photophobia or giddiness; whether he is in the habit of screaming out suddenly or sighing heavily or grinding his teeth; whether he vomits or is constipated, and if he has had convulsions.

In looking for physical signs we must not omit to notice the physiognomy, the condition of the fontanelle and of the superficial cranial veins, and to observe if the pulse is irregular. The state of the pupils, and the presence of squint or nystagmus, of cervical rigidity or head-retraction, are also important.

Defects of ordinary sensibility are less common in children than in adults. Pain on movement of a limb is important, but is not often due to disease of the nervous system.

In investigating any apparent loss of muscular power in a child, we have, *firstly*, to decide whether it is a true paralysis resulting from the pain which movement causes, from extreme flabbiness of the muscles, or from some malformation; and, *secondly*, if a true paralysis, whether it is due to a lesion of the brain, cord, or peripheral structures, or is a so-called functional palsy such as may be met with in hysteria or as the result of peripheral irritation.

Tremor is a rare symptom in childhood, but choreiform or athetoid movements are commoner than in later life.

When it is important to ascertain the condition of the electrical reaction in a young child, it is generally advisable to give an anæsthetic.

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Chloral Hydrat. *See* TOXICOLOGY.

Chloroform. *See* ANÆSTHETICS.

Chlorosis.

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Introductory.—Chlorosis or green-sickness is a disease of the female sex, occurring usually for the first time between the ages of fourteen and twenty years, and having anæmia as its cardinal symptom. The anæmia is due to defective blood formation; it occurs spontaneously, or at least without any cause which is universally admitted to be sufficient, develops rapidly as a rule, and gives rise secondarily to a number of other symptoms. If the disease is not treated it does not tend to recover, but to become more severe or chronic, and even when efficiently treated it is apt to recur. As the patient advances in years this tendency becomes less marked.

Causation and Pathology.—(1) *Predisposing Causes.*—So far as is known chlorosis occurs only in the female sex, and the *first* attack usually falls in the years between fourteen and twenty, very seldom earlier and seldom later. Recurrences may occur at any time after the first attack, and have been met with comparatively late in life. The disease often runs in families, especially in large families, and the mothers of chlorotic girls have themselves often been affected. No other illnesses in the parents have any special relation to chlorosis in the children. There must be a predisposing weakness of a functional character in the blood-forming organs, but Virchow's belief that the cause of the disease is a general hypoplasia of the vascular system, and the kindred notion that it is due to a hypoplasia of the genital organs, are certainly erroneous. It is impossible that a disease so curable should depend on organic defects so serious.

(2) *Exciting Causes.*—Such bad hygienic conditions as want of fresh air and of light in the rooms which patients inhabit, and such faults in clothing as corsets so tight as to interfere with the proper action of the viscera, may assist in producing the disease. An improper amount of work and exercise is probably more important, and more harm is done by too much fatigue than by too little. Unwisdom in the choice of food or inability to obtain a proper quantity or quality is even more serious. The iron of the blood is normally regenerated from the iron of the food, though there is a reserve in the liver which can be drawn upon in emergency. Normally the intake of iron in the food, in men at least, just balances the output, but young women tend to prefer other foods to the iron-containing proteids of a man's diet, and their intake of iron is thus often insufficient. Moreover, chlorosis is very apt to appear at the time when girls have left school and home, and have begun to work. They need a fuller diet, especially more proteid food, and often cannot afford it, are not allowed it, or will not take it. Bad hygienic conditions, dyspepsia, constipation, mental depression, all lead to loss of appetite and to a diminution in the amount of food taken. In addition to a diminished intake of iron there is in young girls an excessive output. The blood-loss of menstruation means on each occasion an output of iron equal to that ingested in the food in a week, and it must require a healthy appetite and vigorous digestion to make good the loss. Many chlorotics have at one time suffered from menorrhagia, though when the disease is fully established the majority either cease to menstruate, or do so in diminished quantity. In the chlorotic years the

development of the whole body goes on rapidly, and must throw a great strain on the blood-forming organs, and if these are imperfectly nourished, and exposed to loss as well, chlorosis may well result. Very little is known of the mechanism by which the blood-forming organs are stimulated to make good any loss, and it is possible that this may depend to a certain extent on the internal secretion of one or more organs, in the female sex possibly the genital organs. A loss of this stimulation from functional disturbance of the generative organs may endanger the normal course of blood formation.

The view that chlorosis is dependent on constipation has been abandoned. Constipation is not more common nor more severe among chlorotics than among other women, and purgatives alone will not cure the disease. Nor is there ground for believing that an abnormal amount of intestinal putrefaction is present to produce toxins which destroy the blood or hinder its formation. The ordinary evidences in the urine of increased intestinal decomposition are wanting in chlorosis, nor is there any reason to think that excessive blood destruction occurs.

(3) *Pathology.*—Chlorosis is not a fatal disease, and hence little or nothing is known of its morbid anatomy. Fatty degeneration of the heart and other viscera has been found in this as in other anæmias, the stomach may be dilated, and so may the heart. The only change which is characteristic is the condition of the *blood*, and the only alteration there which is absolutely constant is the diminution of hæmoglobin, not only absolutely, but also relatively to the red corpuscles. The red corpuscles in the cubic millimetre vary greatly in number. In cases which are taken into hospital they are often diminished, though very rarely below two million. These, of course, are the severe cases. In out-patient and private practice the number is more usually about three or four million, and not infrequently the number is normal—about 4,500,000—or above the normal. But even in the cases with a normal number of corpuscles the hæmoglobin will often be diminished to 50 per cent—a colour-index of 0·5—while when the corpuscles are unusually low the hæmoglobin also sinks. I have taken 313 cases, tabulated by Thayer, Cabot, Bramwell, and Stockman, and find the average number of red corpuscles to have been 3,800,000, while the average percentage of hæmoglobin was 40·3. This gives a colour-index of 0·47.

The blood when drawn is pale and very fluid, but coagulates rapidly, though the fibrin is not increased. Blood-plates are almost always increased in number; indeed, sometimes in stained films they seem to be almost as numerous as the red cells.

The red corpuscles are small, and when stained are pale, and show specially a much paler centre than usual. This of course corresponds with their individual poverty in hæmoglobin. In slight cases their shape is often otherwise normal, but in severe cases poikilocytosis may be quite as marked as in pernicious anæmia, though the deformed corpuscles still show the central pallor. Nucleated red corpuscles are extremely rare, and are only found in the severest cases. Leucocytosis may occur in chlorosis from any of the causes which ordinarily produce it, but in uncomplicated cases the leucocytes tend to be scanty in number rather than increased, and to be fewer in the worst cases than in the milder ones. The percentage of lymphocytes is typically increased. The specific gravity of the blood and the dry residue are diminished parallel to the hæmoglobin, but the plasma is unaltered. If a case be watched through its development and recovery it will be found that at first the red corpuscles are normal in number, but they become small in size and the hæmoglobin is diminished. Then the number of the corpuscles begins to fall and poikilocytosis appears. As the case improves under treatment the corpuscles increase rapidly in number, but remain for a long time small and pale, and the normal number has been regained long before the corpuscles attain their normal size, and before the hæmoglobin reaches the normal.

The essence of the disease is insufficient blood formation; one might indeed define it as a lassitude of the bone marrow. The demand of the body for fresh corpuscles is complied with, but the corpuscles are small and of light weight, and the oxidation processes in the body are thus interfered with.

Symptoms.—The first complaint is usually breathlessness on exertion, then follow fatigue and palpitation. Pallor, which may be greenish in tint, does not usually appear till the blood changes are well advanced, and shows itself first in the lips and conjunctivæ, later in the skin. The patients often, indeed, have a fresh pink and white complexion, for the vascular nerves are easily excited and flushing results. There is disturbance of menstruation, usually greater the younger the patient and the more seldom she has menstruated before. Complete or partial amenorrhœa is the rule, menorrhagia is much less common. This suppression of menstruation is of course a curative endeavour. Leucorrhœa is common. The muscular power diminishes, though here there are great individual differences due rather to temperament and will-power than to bodily conformation. Headache is common, and in bad cases one meets also tinnitus, temporary blindness, and deafness, giddiness, and fainting. The last is specially apt to occur after the patient has been standing for a long time.

The amount of alimentary disturbance varies greatly. The stomach is sometimes dilated, and gastric ulcer is a not infrequent complication. Some patients retain a normal appetite, and these tend to become fat; others lose their appetite, suffer from dyspeptic symptoms, and become thin. But even those patients who retain their appetite are very apt to be capricious in their choice of food; meat they generally dislike, while they have a special fondness for acids, such as lemons and vinegar, or for such things as chalk, starch, dry oatmeal, dry tea leaves, or dry sago. Constipation is common. The tongue is sometimes furred and flabby, but more usually pale and clean. The pulse is generally rapid; the heart may be enlarged, usually more to the right side than the left. Coldness of the hands and feet or of the whole body, from feebleness of the peripheral circulation, is often complained of, and patients often suffer from "dead fingers" for the same reason. Thrombosis of the veins, especially of the legs, occasionally occurs, and without this there may in severe cases be some œdema of the ankles. It is doubtful whether these thromboses are due to the increase in blood plates or not. In the majority of cases they seem to be organismal in origin. The splenic dulness is often enlarged, but the organ is rarely palpable. The urine is copious, pale, and of low specific gravity.

The patients are often irritable, capricious, or obstinate. The headache may be constant, or may occur in attacks like migraine. Neuralgias of all kinds are very common, especially inframammary neuralgia. Optic neuritis sometimes occurs. The temperature seldom rises unless for some complication.

Cardiac and Vascular Murmurs.—The *cardiac murmurs* of uncomplicated chlorosis are always systolic in time, and are heard most frequently in (1) the pulmonary area, with the point of maximum intensity in the second left interspace, close to the sternum, *i.e.* just over the pulmonary artery. The causation of this murmur has been much discussed. It has been ascribed to mitral regurgitation, to pressure on the pulmonary artery by the dilated left auricle, to wateriness of the blood, and to other causes. The view now most commonly held is that it is due to a want of tone in the walls of the artery, similar to that found in the heart walls, and caused by malnutrition, and that the murmur is produced by the sudden propulsion of the blood into the artery, which is relatively dilated in comparison to its orifice. The same cause would account for the murmur which is sometimes heard in (2) the aortic area, though with much less frequency. Systolic murmurs in (3) the tricuspid area and (4) the mitral area are usually heard along with the basal murmurs, but sometimes alone, and in cases where they are not due simply to the conduction of very loud basal murmurs, are more serious, for they are found only in severe cases of chlorosis, and indicate that the heart wall is so enfeebled as to allow of sufficient dilatation to cause respectively tricuspid or mitral regurgitation. It is sometimes difficult to be certain in these cases whether the murmur is due entirely to chlorotic dilatation or to pre-existing valvular disease. The history and the blood examination generally help us, and the result of treatment usually removes all doubt, as chlorotic dilatation is eminently curable.

Arterial murmurs are sometimes heard at a distance from the heart, but not with sufficient constancy to be characteristic. The *venous murmurs* are heard most easily in the jugular veins, at the root of the neck, especially on the right side. This *bruit de diable*, or venous hum, is continuous, though it differs in intensity from various causes. Its causation is usually ascribed to the fact that, while the upper part of the vein is free, and able to collapse if ill-filled, or if it shares in the general want of tone of the vascular system, the lower part is kept distended by its attachments to the cervical fascia. In the passage of the blood from a narrower to a wider space we have one of the typical conditions for the production of a murmur, which is continuous because of the continuous flow of blood in the veins. A murmur of the same character may sometimes be heard over the eyeball, over the occipital protuberance, and elsewhere. The venous hum, though not confined to chlorosis, is very characteristic of it.

Pulsation is often to be seen and felt in the episternal notch, in the pulmonary area, over the area of the right ventricle, and in the epigastrium. From what has been said of the state of the heart and vessels its causation in each case will be evident.

Course and Prognosis.—Cases vary very much in their rapidity of onset. In some the disease develops in a few days, but in the great majority its incipient stages extend over two or three weeks, or even over a much longer period. Generally speaking, cases with an acute commencement recover quickly; those with a chronic commencement take much longer to get well. But all cases, acute or chronic, show a marked tendency to recurrence. This is largely to be explained by the fact that it is very difficult to persuade chlorotics to persevere with treatment until they have thoroughly regained their health. They are accustomed to a condition of health which is short of robustness, and there is often so marked an improvement after a short course of iron that they are satisfied with it, and drift out of observation. I have seen patients with a hæmoglobin percentage of 50 declare themselves quite recovered, and resent the idea of further treatment. The result is a recurrence in a short time, and these cases often become quite chronic, for relapses do not yield so well to iron as primary attacks. On the other hand, patients who will submit to treatment usually recover completely, and are not nearly so liable to relapses as imperfectly recovered cases. It is, of course, of great importance to impress upon patients the necessity of complete recovery, for patients suffering from chlorosis are in a poor condition to resist acute intercurrent diseases; they are liable to the development of gastric ulcer, and if the disease is allowed to become chronic, though the anæmia may pass off in after years, it leaves its mark in a state of weakened vitality, and often leads to a condition of chronic invalidism.

Diagnosis.—The points on which stress should be laid are—the sex and age, the apparently causeless development of the disease, the history and general appearance of the patient, the character of the anæmia on examination of the blood, especially the disproportionate diminution of hæmoglobin, and the success of treatment with iron. Difficulty may arise in cases where one or other set of symptoms of the disease is unusually prominent; the gastric and cardiac symptoms, and those associated with the generative organs, being most likely to cause error, and it is of course not unusual to find chlorosis in patients who are suffering from organic heart disease, from tuberculosis, or other chronic maladies. From other conditions causing anæmia the differential diagnosis should be made on the following lines:—

(1) *Pernicious Anæmia* (see "Anæmia, Pernicious").—It is comparatively seldom that it is necessary to make this diagnosis, as it is very rarely that the anæmia in chlorosis is so grave as to give rise to the suspicion of the other disease.

(2) *Leucocythæmia.*—The examination of the blood at once removes doubt.

(3) *Anæmia from Intestinal Parasites.*—The type of anæmia is rather that of pernicious anæmia. Where the blood examination leaves any doubt the fæces should be examined for the eggs of the parasites.

(4) *Anæmia from Malignant Disease.*—The type of anæmia is very often chlorotic in malignant disease; but though, of course, malignant disease is not specially common in young girls without causing definite symptoms, it may occur, and sometimes does give rise to difficulty. Leucocytosis is much more common in malignant disease, nucleated red corpuscles are more common and more numerous than in chlorosis, and the effect of treatment usually clears up the difficulty.

(5) *Tuberculosis.*—Nothing is more common than to find early cases of phthisis and other forms of tuberculosis taken for chlorosis, because the lungs, etc., have not been examined with sufficient care. The blood examination gives very similar results, and careful investigation of the history, repeated examination of the sputum, lungs, glands, etc., must be made. The temperature is not always helpful, for early tubercle does not always cause fever, and in chlorosis the temperature is sometimes raised. In very difficult or important cases it might be possible to make the diagnosis by the injection of tuberculin, when reaction or its absence would make the diagnosis easy. It is to be remembered that tubercular tumour of the brain or meningitis may cause anæmia.

(6) *Simple anæmia* from bad hygiene, want of food, of sleep, of light, of fresh air, from overwork, etc.—In these cases the hæmoglobin and red corpuscles are more likely to be equally diminished, and the blood-plasma also becomes less albuminous; but it is only in the extreme cases that this holds, and it is necessary to inquire very carefully into the patient's history. No amount of iron will take the place of sleep or fresh air in treatment.

(7) *Kidney Disease.*—Chronic nephritis always causes anæmia in young people, but the blood-plasma contains a smaller amount of solids. The examination of the urine usually clears up the diagnosis.

(8) *Anæmia from Chronic concealed Hæmorrhage.*—This may sometimes be from piles, of which the patient, from ignorance or modesty, does not tell her doctor, but more often from ulcer of the stomach or duodenum. These ulcers may give rise to no symptoms, or to none that are not usual in chlorosis, and the blood in the stools is so much altered that the lay public cannot recognise it. The blood examination gives little or no help here, and there will be need of a very careful review of the whole case, and repeated examination of the stools.

(9) *Early Pregnancy.*—This must always be borne in mind as a possible cause of amenorrhea and anæmia in young girls.

TREATMENT.—(1) *Prophylactic.*—In families where chlorosis has occurred in the elder sisters, it is worth while to be specially careful of the health of the younger sisters during the years when they are likely to be attacked. From what has been said with regard to the etiology of the disease it will be evident what the necessary measures are—a healthy life, with sufficient food, air, and exercise. It is quite useless to give iron before the disease develops, as it does not prevent the onset of chlorosis, and, if the system has become habituated to its use, it may fail entirely to assist blood formation when it is really needed.

(2) *General.*—Sunlight and fresh air are of prime importance, and too much attention cannot be bestowed on them. Rest is also an all-important factor. All severe cases should be sent to bed, and so should all cases of medium intensity whose circumstances will allow of it. This removes the strain on the enfeebled and badly-nourished heart, and the symptoms of dyspnœa, faintness, headache, and neuralgia are relieved almost immediately. The length of time that the patient is to remain in bed depends on the severity of the case and the success of treatment; generally three weeks is a sufficiently long time, but it should be some weeks longer before she is allowed to resume her ordinary duties. In slight cases, where it is not necessary to make the patient go to bed, she should rest as much as possible, avoid fatigue and excitement of all kinds, and keep early hours.

The diet should be regulated to suit each case, of course; but regularity of meal times should be insisted on, and the want of appetite and disinclination for food can often best be dealt with by ordering relatively small meals at shorter intervals than usual—say every three hours. The distaste for meat which almost all chlorotics evince must be overcome, gently but firmly; and an ordered quantity of it, small at first, but increasing, must be taken. The previous diet has often consisted of bulky but innutritious foods, and these should be replaced by proteids, whose concentrated nourishment can be more rapidly utilised. When the patient is thin, milk with an equal quantity of cream added to it may be given in addition to the meat, and where patients cannot take solid food milk will be given largely; but to fat patients, and to those who have a good appetite for solid food, a large quantity of milk should not be given. Alcohol, which favours fat formation, may be given to thin patients. The popular superstition that claret and other red wines "make blood" is of course absolutely groundless. Cold bathing, or anything else which withdraws heat or causes shock to the heart, should be avoided. The bowels must be carefully regulated, as iron has a tendency to produce constipation, though this is often overrated.

(3) *Special.*—Iron cures chlorosis in the great majority of cases. It does not do so by replacing lost or diminished iron in the blood. The iron-containing proteids of the food are quite capable of doing this, but rest and good food alone will not cure chlorosis. What is necessary is a very active stimulation of the bone-marrow, and the salts of iron circulating in the blood are the best stimulant to the marrow. The form in which iron is to be taken is to a certain extent a matter of indifference, and depends upon the digestive powers of the patient, our ability to give a sufficient amount of iron without giving too bulky a dose of the preparation, the experience of the physician with similar cases, and only secondarily on the special properties of individual iron preparations. All of them, organic and inorganic, are transformed in the stomach into ferric chloride. It was

supposed that the inorganic salts of iron were not absorbed, and as a result there were put upon the market numerous preparations of iron-containing nucleo-albumins and proteids, under the names of hæmoferrum, hæmol, hæmatogen, hæmoglobin, carniferrin, ferratin, etc. These contain iron in organic combination, and were supposed to be more easily assimilable than the inorganic salts. They are all, however, too much like food-iron, do not stimulate the marrow with the same rapidity or vigour as the inorganic salts, and many of them have the further disadvantage that they contain so small an amount of iron that enormous doses would have to be taken to make up the necessary minimum daily dose, which is from 0·1 to 0·2 grammes of metallic iron. Of the inorganic salts, which we now know can be absorbed, the one which is most used is the carbonate, in the form of Blaud's pill or capsules, or as the saccharated carbonate. It is not astringent, and is generally well taken. The protosulphate and the ferric salts are astringent, and cannot usually be taken by people with irritable stomachs, but where the digestion is good they are often very useful. Best of all, perhaps, is reduced iron, provided it does not contain sulphur as an impurity; when it is dissolved in the gastric juice hydrogen is evolved, and if sulphur be present, unpleasant eructations of sulphuretted hydrogen result. Its small dose is an advantage. The scale preparations are sometimes useful with dyspeptics, as they are easily taken, but they contain a relatively small amount of iron.

The dosage should be carefully regulated. Blaud's pill may be taken as a standard, and of these six a day, two after each meal, should at first be given. After three or four days or a week the number may be doubled, if there is no increase of gastric disturbance, and after another week the number may again be increased. It is rarely necessary to give more than from 20 to 24 pills a day, but the maximum, when once reached, should be persevered with for some weeks, and the dose then gradually decreased for a fortnight before leaving off altogether. The length of time during which the iron is to be taken varies in different cases, and should be determined by the effect on the blood. As long as the hæmoglobin percentage improves the iron should be continued, but if it becomes stationary it is best to stop the iron, let the patient go without any for from two to four weeks, and then begin again in the same way. It is generally a mistake to let a patient go on taking iron indefinitely, as the system becomes habituated to its use, and if a relapse occurs treatment is not so effectual. Iron must always be given after food; no matter what form is taken, it must be taken regularly and without interruptions, and as the dyspepsia of chlorotics is usually due to their anæmia, iron should be given even though the patient complains of slight dyspeptic symptoms. It may sometimes be necessary to give a bitter tonic, or some such remedy as bismuth, rhubarb, and soda before food for a few days, while the iron is being given, and it is often wise to begin treatment with a purge.

In cases where the iron is apparently doing no good it is well to see that the preparation which is being taken is really active, to see, for instance, that the pills are soluble, or to change the preparation. Chalybeate waters are sometimes useful in such cases, or arsenic may be tried along with or instead of the iron. It is most likely to be useful in cases where the number of blood corpuscles is greatly reduced, and it must be given at first in small doses and gradually increased. Blood-letting has been used as a means of treatment, and might be tried in those very chronic cases where drugs are not very successful. Hæmorrhage is a powerful stimulant to the marrow, and may give the necessary fillip to blood regeneration which can then be maintained by iron. About four ounces of blood might be drawn.

Treatment should in all cases be continued until the normal hæmoglobin percentage is reached, and it must be remembered that patients often recover a healthy appearance long before this result is attained.

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Cholelithiasis. *See* GALL BLADDER.

Cholera, Epidemic.

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SYNONYMS.—*Asiatic cholera*; Fr. *choléra*; Ger. *Brechruhr, Gallensucht*; It. *colera asiatico*. The Indian names are *morshi, mordeshin, visuchika* or *bisuche, haiza, sitanga, waba*, etc.

DERIVATION.—The word cholera was applied by the Greek and Roman physicians to the disease known as summer cholera or cholera nostras. Its etymology is uncertain. Celsus supposed it to be derived from χολή, bile, and ῥέω, to flow; others, with Alexander of Tralles, derive it from χολάδες, the intestine, and ῥέω, to flow, while some recent authorities incline to the view that it comes from χολέρα, the gutter of a roof.

BACTERIOLOGY.—In 1884 Koch discovered the comma bacillus or vibrio, which is now generally recognised as the *ens morbi* of cholera. Pure cultures of the microbe are undoubtedly capable of giving rise to the disease in man; and if the evidence derived from the experimental production of the cholera in the lower animals is more ambiguous, this arises from the fact that cholera is essentially a human disease, which does not give rise to the same train of symptoms in the lower animals that it does in man. So much unanimity now obtains respecting the pathogenic character of the vibrio, that it is unnecessary to recount the accidents and experiments which establish beyond doubt that all the symptoms and lesions of cholera can be caused by it in man, or to discuss the value of the evidence afforded by experiments on the lower animals.

The vibrio is found in the stools of cholera patients, and after death in the contents and tissues of the intestinal canal. It has occasionally been detected in the vomited matters, but only in small numbers, and its presence in the vomit is doubtless to be accounted for by the contents of the intestine finding their way into the stomach. It has also been met with in a few instances in the bile-ducts and gall-bladder. It is never present in the blood, liver, spleen, kidneys, or mesenteric glands. The real seat of the microbe is the lower part of the small intestine. Abel and Claussen made the important observation that vibrios are often present in the stools of healthy persons who are in daily intercourse with cholera patients. They have also been found in the stools of convalescents up to fifty days after recovery.

When one of the small mucous flakes from a cholera stool is spread out on a cover glass, dried, heated, and stained, the bacillus is readily to be seen if the specimen happens to be a pretty pure cultivation, otherwise the presence of other organisms makes its detection difficult. In many cases its presence or absence can only be certainly determined by placing a mucous flake in peptone broth, and incubating for twenty-four hours; the vibrios in sufficiently pure culture for microscopic examination are then to be found on the surface of the fluid. The various media and processes of cultivation, and the distinctive characters of cholera colonies, will be found described in any text-book on bacteriology.

The cholera bacilli are actively motile, flagellated, curved rods, about half the size of the tubercle bacillus. Their average length is about 1·5 μ; their thickness from one-sixth to one-third their length. The young forms show only a slight curve, the older ones a more decided bend, while some present the form of a half circle. They occur isolated or attached to one another in the form of the letter S, or in longer screw-like forms. They are stained with warm solutions of methylene blue, or with methyl-violet, or fuchsine.

They do not thrive in acid media. They are ærobic, but are nevertheless capable of growing to some extent when oxygen is altogether excluded. The cultivations made with a deficient supply of oxygen are more virulent, but less resistant to the action of the gastric juice and external influences than those grown with a more abundant supply of oxygen. It is the circumstance that they are capable of growing when only a slight amount of oxygen is present that explains their rapid multiplication in the intestinal canal. This may also explain their exalted toxic power as parasites, a toxicity which they lose when grown for a time as saprophytes with a free supply of oxygen.

The cholera bacillus does not grow below 16°; it thrives between 22° and 25°; its optimum lies between 30° and 40° C. It is killed by exposure for half an hour to a temperature of 60°. Although the bacillus does not grow below 16°, Koch found that it is not killed by being subjected for an hour to a temperature of $-10°$ C. It is very susceptible to drying. Koch says that when spread out on a cover-glass and exposed to the air, the bacillus is killed after two or three hours; but according to Kanthack the vibrios have been found to retain their vitality for 120 days when dried on glass. When exposed to sun and air they do not live long on a cover-glass, but if the air is humid, and the preparation is not exposed to sunlight, they are not so easily destroyed.

Commas are easily destroyed by the growth of saprophytes, but experiments bearing on this point give widely different results. The nature of the medium, its reaction, and the class of organisms it contains all no doubt count for much. Koch found that when added to sewage the vibrios could not be demonstrated after twenty-four hours; on the other hand, they have been observed to retain their vitality for months in sewage-polluted water. Koch did not find them to survive longer than six or seven days in the water of the Berlin Canal. Örgel found that they could live for nearly twelve months in ordinary Elbe water. They grow at first very luxuriantly in moist soil, but after a few days they generally succumb to saprophytes. They retain their vitality for a long time on moist linen, on which they are often found in a state of pure culture. The vibrio is not very fastidious as regards food. It grows on agar-agar, to which meat infusion and peptone have been added, in slightly alkaline nutrient gelatine, on solidified blood serum, on potatoes, milk, and, to some slight extent, even in sterilised water. This shows its adaptation to saprophytic growth.

So far we have been dealing with the characters and habits of the microbe as observed in experiments. Little is known of its life history outside the human body, but what little we know shows that it can retain its vitality longer than cultivation experiments would lead us to anticipate. The microbe under natural conditions is a hardier plant than many suppose. In the sewage-polluted water of Marseilles harbour it has been found to survive for eighty-one days. There is reason to believe that it may exist not for the few days which experiments indicate for its limit, but for months, perhaps for years, in the soil (see article on "Epidemiology").

But while the cholera vibrio is capable of retaining its vitality, it loses much of its virulence when grown for a time as a saprophyte, and the more vigorous its saprophytic growth, the less its virulence. From Hankin's observations this loss of virulence is observed even in India, where the conditions might be supposed to be peculiarly favourable for it retaining its properties. Not only does it lose in virulence when grown continuously outside the body, but it also undergoes marked morphological changes.

It is admitted that the vibrios met with in cholera stools present considerable differences, which tend to perpetuate themselves through successive generations. Some have one flagellum, others more. Less definite distinctions in size and form are also observed. Differences, too, exist in the appearances presented by the colonies they form on gelatine plates, and in their power of liquefying gelatine, and all this has given rise to discussions as to the unity or multiplicity of the germ. The marked morphological and biological changes in vibrios, derived from a common stock, which are observed to occur under cultivation, prove the variability of the organism, but the essential unity of all the varieties is attested by their common pathogenic properties, and by the immunity which the vaccine of one variety confers from attacks of all the others.

When the cholera vibrio is cultivated in the peritoneal cavity of the guinea-pig its virulence is increased, and it is by passing it in this way through a succession of guinea-pigs that Haffkine obtains his prophylactic virus. A culture from the peritoneum furnishes a pure sub-culture on agar, which is thoroughly shaken up with broth. This constitutes the vaccinating fluid, which may be used as a living vaccine, or the bacillus may be killed before being used.

Very great interest and importance attaches to the investigations of Metschnikoff bearing on the influence of non-pathogenic organisms in promoting or hindering the development of the cholera vibrio on external media, and in determining infection in animals. He found that some species of sarcinæ, torulæ, and a variety of the colon bacillus promoted the infection of suckling rabbits. The associated organisms rapidly disappeared, but none the less they subserved to the development of the cholera virus in these animals. When we remember the effect of moulds in promoting the growth of the bacillus of yellow fever, and of streptococci in intensifying the virulence of the diphtheria bacillus, we feel that it is in this association of non-pathogenic organism with the cholera bacillus that we are likely to find an explanation of some obscure points in the etiology and epidemiology of the disease.

The manner in which the comma bacillus is influenced by temperature, moisture, rainfall, and drought throws considerable light on some of the epidemic features of cholera

(*a*) The relation of the vibrio to temperature explains why in temperate climates cholera epidemics are mostly restricted to summer and autumn. It is in these seasons only that the temperature permits its saprophytic growth.

(*b*) The effect of drying on the vitality of the parasite accounts for cholera dying out during the long dry season in the Punjab and Central Provinces, and its reappearance after the rains begin to fall, when the earth and atmosphere become moist and humid. In such regions, the cholera season is regulated not by the temperature as in Europe, but by the rains.

(*c*) The inability of the vibrio to live for any length of time in fluids deficient in nutritive material, as well as the effect of excessive moisture of the soil in reducing the oxygen at the disposal of the organism, explain the subsidence of the disease in endemic areas, when the heavy rains submerge large tracts of land, and displace the air from the soil that remains uncovered.

ETIOLOGY.—For a widespread epidemy of cholera the following conditions are necessary:—(1) The presence of the microbe; (2) a suitable medium and temperature for its growth outside the body; (3) means of transport from place to place; (4) a vehicle by which it can be diffused in a particular locality; (5) a susceptibility for infection on the part of a community.

1. *Sources of the Virus.*—The primary source of the virus, outside the regions in which cholera is endemic, is in every instance the intestinal discharges of a person suffering from, or one who has recently suffered from the disease, or of a person who without suffering from the disease harbours the microbe, from having associated with a cholera patient.

It will seldom happen, however, that the microbe grown in the intestinal canal of a cholera patient will find its way directly into that of a healthy person, for cholera discharges will only be swallowed accidentally, or as the result of a scientific experiment. It is the descendants of this microbe grown in some external medium—water, milk, soil, linen, which in most cases causes infection.

2. *Breeding-places.*—The virus having been introduced into a locality must find some medium outside man in which it can grow. Such media are (*a*) a soil polluted with organic matters, especially excreta; under soils we include accumulations of animal and vegetable refuse, cesspools, etc.; (*b*) sewage-polluted water; (*c*) milk and other articles of food.

Having found a suitable medium, the bacillus requires a certain temperature for its growth. Cholera was introduced simultaneously into New York and New Orleans in December 1846, and broke out in both cities; but in New York the outbreak rapidly subsided, whereas in New Orleans it spread during the winter. This difference will be easily understood when we remember that the winter temperature of New York is under freezing-point, while that of New Orleans is about 60° F. How favourable soever other circumstances may be, an extensive epidemy of cholera cannot occur in winter in higher latitudes, except under very exceptional conditions.

3. *Means of Transport.*—The view that the cholera virus can be transported for long distances by the air is quite untenable. Bryden maintained that atmospheric moisture is the carrier of the virus, and that its distribution over India extends as far as the moisture-laden monsoon winds carry it. The rains supplied by the monsoon act in a different way; they bring the soil into a condition fitted for the spread of the pestilence. The germs of the disease are always being carried from Bengal to the north-west, but they do not develop epidemic outbreaks until the condition of the soil favours the growth of the microbe.

The principal means by which the virus is transported from place to place, so as to overrun vast regions, are (*a*) *human intercourse*, that is, by persons who have, or have had, the disease, or who harbour the vibrio, and things contaminated by cholera discharges. That this is the principal means of its propagation is proved by the constancy with which cholera has followed caravan routes and lines of communication by river, road, rail, or ship, and by its rapidity of spread increasing as intercourse between distant countries becomes more rapid. In its early invasions of Europe, cholera followed the routes of caravan traffic. Cabul has always received the infection from India, and has been the centre from which it has advanced westwards. One route from Cabul passed through Central Asia, by Balkh, Bokhara, and Khiva to Orenburg; another through Persia by Herat, Mesched, Astrabad, Teheran, Reshed, Baku, and Astrakhan. An alternative route from Teheran led through Tabrees, Tiflis, Erzeroom, and Trebezond to the Black Sea. In the same way cholera invariably breaks out in an island at ports in communication with an infected place. In Russia in the olden time cholera followed the rivers, and on its first outbreak in England in 1832, it followed, as Hirsch points out, " the commercial highways chiefly, and the coast routes and rivers, while the mountainous parts of the country were little visited by it, and the Scottish Highlands not at all."

When we remember that cholera has been five times epidemic in Mauritius, and that on each occasion it has broken out shortly after the arrival of vessels from India with the disease on board; that it broke out at Quebec in 1832 four days after the arrival of the *Carrick*, on board which cholera had prevailed during the voyage (and the western hemisphere had never up to that day been visited by the disease); that it appeared at New York in 1848, which was then free from the infection, on the arrival of a vessel which had lost seven passengers from cholera; that it appeared at New Orleans in the same year, three days after the arrival of the ship *Swanton*, thirteen of whose passengers had died of cholera during the passage; and when we see it breaking out along the routes of pilgrims, tracking the march of armies, following the lines of emigration, we will be driven to the conclusion that human intercourse is the most important of all the means by which cholera is transported from place to place, from one country to another, across deserts and oceans.

(*b*) *Rivers.*—A stream polluted by cholera discharges may carry the virus for very considerable distances to towns situated on its banks. In the last outbreak in Germany the frontier river Przemsza, a tributary of the Vistula, had become polluted by the cholera virus. Shortly afterward, cases began to appear among the river raftsmen and bargemen on the Vistula. The cholera bacillus was found in the water of the river above Dantzig, where cases now began to appear. By the middle of June at least six German towns and villages on the banks of the river had become infected (see *Local Government Report*, 1897-98).

(*c*) *Ships*, which are moving centres of infection, transport the virus across oceans. This usually happens through the agency of infected persons, but it may also take place by means of infected goods, ballast, or bilge water.

3. The diffusion of cholera in a given locality is a question distinct from that of its transport.

(*a*) The vehicle by which it is most frequently conveyed into the system is *drinking water*. When the virus finds its way into the general water-supply of a city, as was the case in Hamburg in 1892, the disease becomes rapidly and widely diffused. When wells become polluted we have local outbreaks, as happened in the well-known Broad Street explosion in London in 1854, in which the disease was practically confined to those who made use of a contaminated well, and ceased from the day on which the well was closed.

The important part played by water in the spread of cholera is seen in the decrease of the disease in towns in which it was formerly prevalent on the introduction of a pure water-supply. The average mortality of the European troops at Fort-William, Calcutta, was 20 per 1000 from 1826 to 1863. From the latter date, when the fort was for the first time supplied with pure water, to the present time it has averaged 1 per 1000.

Lahore had an average cholera death-rate of 1·07 per 1000 for the fifteen years 1848-61. In the period 1882-87—that is, after the introduction of water into the city from the Ravi River—it fell to 0·07 per 1000. that the reduction in the cholera death-rate has really been due to the improved water-supply is proved by the fact that in the Lahore district (excluding the city) the cholera mortality, which was 0·34 in the former series of years, rose in the latter period to 0·43 per 1000.

(*b*) *Milk and other articles of food* are not only media for the growth of the virus, but vehicles for its diffusion. Insects doubtless play an important part in spreading the disease by settling on articles of food after having been in contact with substances containing the virus. The house-fly in particular is an active agent in disseminating the germs of the disease in this way, and now that it has been shown that the vibrio is capable of living for at least fourteen days in the fly, wider limits must be assigned to its pernicious activity than was formerly conceded to it.

(*c*) *Air as a Vehicle of Infection.*—Hirsch, writing in 1883, says, "The facts do not permit us to ignore that the poison *must* be taken up and suspended in the air, so as to enter the human organism with the breath." This was how the facts looked to so eminent an epidemiologist a few years ago. The facts remain the same, but the way in which they are looked at and interpreted has so completely changed, that to many it seems now incredible that the virus ever enters the system by means of the breathing air. It is held that micro-organisms can only be detached from perfectly dry surfaces, so as to be carried about with dust in the atmosphere, and as the cholera vibrio is supposed to be unable to bear this amount of drying, infection through the atmosphere is regarded as impossible. The premises are doubtful, and the conclusion contrary to well-observed facts. That cholera is not usually diffused by means of the air is evident enough, but air is nevertheless one of the vehicles by which the virus enters the economy. It is not to be supposed that infection takes place through the lungs. The vibrio lodged in the upper air-passages may live or even multiply in the alkaline mucosities of the parts, and then be conveyed into the stomach along with food.

As space forbids us entering into detail, we shall content ourselves by briefly referring to two categories of facts illustrating the occasional aerial convection of the cholera virus.

To the first category belong particular instances in which the infection has been carried from the sick-room, and those in which simple proximity to a source of infection has given rise to the disease. Copland records an instance in which all the circumstances seemed to demonstrate that he carried the infection on his clothes for a distance of about a mile and a half and communicated the disease to two of his relatives (*Dictionary*, article "Epidemics"). Similar instances are by no means rare. Simple proximity to a source of infection may also give rise to infection. Cholera broke out on the steamship *England* from Liverpool. There had been 150 cases and 48 deaths on board when she bore up for Halifax. A pilot hailed the vessel, but having learned that there was a fatal disease on board, he laid his boat close alongside, sent up his papers to the captain in a basket that had been lowered from the ship, and brought the vessel up to the quarantine station *without having boarded her*, and then rowed ashore with his two comrades. Two days after having come thus remotely into contact with the *England* he was taken ill of cholera, and three days afterwards cholera broke out in his family. Almost at the same time one of his two companions sickened and gave the disease to three of his children. The Western Hemisphere had then been three years free from cholera (Hirsch). An instance of approach to a dead body being followed by cholera is given by Clemow in the *Transactions of the Epidemiological Society* for 1893-94.

To the second category belong the sudden explosions of a number of cases of cholera on board vessels in which the disease had before been occurring in a sporadic way, after a storm during which the ports and other means of ventilation have had to be closed. These scarcely admit of any other explanation. Instances of this kind are given in detail by Smart.

The *Britannia* ship of war, for example, in 1854 was infected while lying at Buljick, where cholera existed. Up to the 10th of July three deaths had occurred. She put to sea on the 12th, and the disease seemed to subside at once. On the evening of the 13th a gale necessitated the closing of the ports on the sleeping-deck. About 10 A.M. on the 14th "a great and sudden outburst of collapsed cases occurred." There is nothing in the circumstances in such instances (and they are numerous) to incriminate water or food, but everything points to the virus diffused through the air of crowded and unventilated holds causing these sudden explosions.

4. Individual susceptibility counts for much in the matter of infection. It is only a small proportion of a community that is attacked during an epidemy. Excesses of all kinds, especially alcoholic excesses, causing gastro-intestinal catarrh, predispose to cholera. A community acquires an immunity lasting for three or four years after passing through a severe epidemic of cholera.

A few circumstances relating to the etiology of cholera require to be mentioned:—

(*a*) *Topographical Relations.*—Cholera shows a special predilection for the low-lying parts of a town. Farr laid it down as a law "that the proportion of deaths from cholera is inversely as the elevation of the ground," and this law holds good excepting in instances in which the incidence of the disease is determined by the contamination of a water-supply. As an endemic disease, cholera is limited to altitudes not exceeding 1500 feet. As an epidemic malady it has broken out at elevations of 6000 feet (Kussouli, 1845).

(*b*) *Racial Relations.*—The following proportions per cent were attacked at Guadeloupe in 1865:—

Chinese	2·7	Hindoos	3·26
Whites	4·31	Mulattoes	6·31
Negroes	9·44		

The comparative immunity of the Chinese has also been noticed in Mauritius, and has been ascribed to their opium-eating habits, but it is to be remembered at the same time that nowhere has cholera raged more destructively than in the Chinese Empire.

(*c*) *Personal Relations.*—Sex has no influence on the liability to cholera. Its incidence on different ages varies in different outbreaks. In the Hamburg outbreak of 1892 the ages fifteen to twenty-five gave the smallest ratio of attacks and also of deaths to the number attacked. Children under two years of age suffered considerably, as did also old persons. The poor, as a rule, suffer more than the rich, and a special liability attaches to the inmates of Lunatic Asylums. As respects occupations little can be said, except that washerwomen employed in washing cholera linen contract the disease out of all proportion to its incidence on the community generally.

Exempted Places.—Some places seem to be proof against cholera. It has never appeared in Cheltenham, Sedan, or Wurzburg. In other places, such as Versailles, Lyons, and Martinique, the disease has never assumed epidemic proportions.

MORBID ANATOMY

When death occurs at the height of the disease, rigor mortis is well marked; the features are pinched, the face, extremities, and body generally are more or less cyanotic.

The cerebral sinuses and the veins of the meninges contain dark blood.

The pleuræ are dry, and frequently present numerous ecchymoses. The lungs are dry and collapsed, and are much below the normal weight. The larger branches of the pulmonary arteries contain blood; the smaller arteries, capillaries, and pulmonary veins are empty.

The pericardium does not contain a trace of serum. The visceral layer is frequently studded with small ecchymoses. The right side of the heart and the veins emptying into it, as well as the jugular veins, the portal vein, and the larger hepatic veins, are engorged. The left side of the heart is empty and contracted.

The stomach is empty; the lining membrane may be pale and sodden or congested. Occasionally it presents ecchymotic points. The duodenum and jejunum are frequently hyperæmic continuously or in patches.

The large intestine is contracted. The peritoneum covering the small intestine has often a rosy appearance and is dry and sticky. The ileum usually contains more or less rice-water fluid, and is throughout, especially in its lower part, congested and œdematous, but these appearances are most marked in its lower half. Occasionally the mucous membrane is found pale, instead of congested; at other times ecchymosed and suffused. The solitary glands and Peyer's patches are prominent, the latter often surrounded by a red zone of congestion. On microscopic examination the mucous membrane in many parts is found denuded of epithelium. At other places the epithelium is detached from the subjacent tissues by serous exudation. Commas are found in the tubular glands, and between the epithelium and the basement membrane; on the surface of, and sometimes within the villi, and occasionally also in the deeper tissues of the mucosa. The mesenteric glands are swollen and softened.

The liver is often somewhat increased in volume, dark, congested, but of normal consistence. The gall-bladder is full of bile of varying viscidity and colour. The spleen is small, dry, and anæmic.

The kidneys, when death occurs early, are often augmented in volume, and in this case the medullary and cortical substances show punctuated, patchy, or striped areas of venous congestion, occasionally ecchymotic points or patches. The vessels of the glomeruli are congested. In other cases the congestion is less marked, and the kidneys may even be pale. The epithelium of the tubules is swollen and cloudy, blocking up the lumen of the tubes. The bladder is empty and contracted.

When death has occurred during the stage of reaction, the appearances are altogether different from those just described, and the lesions vary considerably according to the symptoms developed during the reaction.

The cerebral membranes are injected, and there is often effusion into the meshes of the pia mater and into the lateral ventricles. The lungs are congested, and the pneumonic or other inflammatory lesions may be present. The mucous membrane of the small intestine may be softened, ulcerated, or covered with patches of diphtheritic exudation, and this may extend to the large intestine. The liver and spleen are usually somewhat enlarged and congested; the kidneys enlarged, vascular, of a dark red colour, with fatty and granular casts in the canaliculi. The urine in the bladder is generally albuminous.

SYMPTOMS

The cardinal symptoms of cholera are diarrhœa, vomiting, muscular cramps, paresis of the heart, algidity, suppression of urine, followed by death or reaction.

The attack may begin with a diarrhœa indistinguishable from an ordinary diarrhœa except by bacteriological examination of the discharges. This premonitory diarrhœa usually lasts from half a day to two days before the choleraic symptoms declare themselves, and when promptly treated, the disease is frequently arrested at this stage. This premonitory stage is often enough absent.

The actual attack begins with frequent, copious, watery evacuations, at first coloured with bile, but soon becoming pale, having an alkaline reaction. Along with this there is urgent thirst, great discomfort in the bowels, an indescribable feeling at once of intolerable distension of the abdomen and sinking that cannot be understood by those who have not experienced it, nor forgotten by those who have. There is no tenesmus.

The stools become more frequent and copious. They flow in streams. No sooner has the patient evacuated, as he thinks, the whole contents of the bowel and hopes to obtain a little respite, than he is again disturbed, and again passes an enormous quantity of an odourless, colourless, rice-water liquid, which on rest, deposits fine, flaky particles.

After a time vomiting sets in, and so much the sooner, the more freely the patient has gratified his craving for water. Large quantities of a pale liquid are ejected forcibly, but without effort, from the mouth. It seems as if it were automatically pumped out. The patient is now extremely restless, tosses off the bed-clothes, and although his skin feels cool or cold, he complains of heat. The pulse is rapid and weak, the heart's action feeble. The extremities become cold and blue, the fingers shrivelled and livid, the temperature in the axilla may fall to 94° or 93° F., sometimes lower; that of the rectum is several degrees above the normal, and it may be well to remark that the rectal temperature is often higher than normal before the algid state has declared itself. It happens, however, in many cases that the rectal temperature follows closely that of the axilla. The voice becomes weak and hoarse—the so-called *vox cholerica;* the eyes are sunken, the eyelids half closed, the face pinched, the breath feels as if it had passed over ice, the tongue gives, as Dr. Watson says, the sensation as if one were touching a frog's belly; the urine is scanty or suppressed. This is the algid stage of cholera.

When this state has begun to develop, or even before, painful muscular cramps of the calves, arms, and sometimes of the abdominal muscles set in, which add greatly to the sufferings of the patient. They are tonic spasms, lasting for two or three minutes.

When the algid state is established, the motions usually become scantier and less frequent, and are passed in bed. Or they may entirely cease, being retained from a paralysed state of the bowel—a symptom of evil omen. Retching, alternating with hiccup, takes the place of the vomiting.

This state may last for a few hours only, or persist for one or even two days—perhaps eight to fifteen hours is the average,—and terminates in reaction or in death.

When the disease is tending to a fatal issue, the patient sinks into an apathetic state, heedless of what is passing around him, but at the same time conscious. The heart's action grows weaker, the pulse imperceptible at the wrist, scarcely to be felt even in the carotid, the skin becomes covered with a clammy sweat, and the patient dies in a state of collapse.

Before the algid stage has set in, or after it has been established, things may take a favourable turn by the setting in of reaction. The pulse becomes fuller, slower; the impulse of the heart stronger; the breathing deeper, easier, and less hurried; the rectal temperature falls, and it is only after this that the surface regains its warmth. The cramps disappear, the retching and purging subside, and the patient falls into a tranquil sleep, from which he awakes refreshed. It is often not until after thirty-six to forty-eight hours that urine is passed. The first urine is of low specific gravity, containing more or less albumin, sometimes blood. In favourable cases the recovery is rapid.

But reaction does not necessarily imply that all danger is over, for in many cases it introduces a new series of troubles. The reaction may be imperfect, the discharges recur, thirst returns, and the patient, when we were beginning to hope for recovery, falls back into the algid stage.

In other cases he lapses into a typhoid condition marked by great debility, more or less stupor, restlessness, especially at night. There is complete anorexia, occasional vomiting, diarrhœa or constipation, or both alternately, with meteorism, scanty albuminous urine, containing fibrinous casts. After this condition has lasted from four to seven days, the symptoms may gradually improve; in this case the appetite returns, the urine becomes more abundant, the albumin diminishes, and the head symptoms pass off.

In other cases the symptoms become aggravated, and the patient falls into a comatose state. It has to be mentioned that typhoid symptoms terminating in death from coma occasionally occur, although the urine is abundant and free from albumin. This typhoid condition is all the more likely to supervene the longer the algid state is prolonged. Those who have been addicted to drink seldom recover without exhibiting typhoid symptoms, which in their case are even more dangerous than in others. Gangrene of the penis, scrotum, nose, and of the mucous membranes of the mouth are the rarer sequels of cholera. More frequently the parotid glands become swollen and inflamed, and sometimes suppurate.

The duration of the disease in fatal cases varies greatly. A few die from four to eight hours after the commencement of the attack, more from twelve to forty-eight hours. The algid state seldom lasts beyond the second day. Those who survive this, die in the typhoid stage from the fourth to the tenth day. After the tenth day recovery is to be expected.

Analysis of Symptoms

Diarrhœa.—The watery, pale stools of cholera are alkaline in reaction, have a specific gravity of 1005 to 1010. The dried deposit from a pint of cholera dejections was found by Parkes to weigh only 4 grains. Examined microscopically, the stools are found to contain intestinal epithelium, dissassociated or in small flakes, with granular matter resulting perhaps from the disintegration of epithelium along with mucous corpuscles. Before the period of reaction the stools will be found to contain flakes of mucus containing commas in almost pure cultivation. In the reaction stage these are less readily to be detected, other micro-organisms then abound, and blood cells, not common in the first stage, are now frequently present.

Chemically, the rice-water evacuations contain a small quantity of albumin, chloride of sodium, and carbonate of ammonium; occasionally, but very seldom, traces of urea.

The diarrhœa is the primary and constant symptom of cholera. In what has been inappropriately called *cholera sicca*, in which there is neither diarrhœa nor vomiting but rapid collapse, the bowels are after death found distended with rice-water fluid, transuded but not evacuated. The bowel in this dangerous form of the malady is paralysed.

Whether the diarrhœa be caused by the presence of the bacillus in the mucous membrane, abstracting from the blood the liquid necessary for its growth, or by a toxin formed by the bacillus, has not been ascertained.

Diarrhœa is only a symptom, it is true, but it is a symptom producing other symptoms, formidable in itself and in its results. The drain of fluid from the system causes inspissation of the blood; this in turn leads to the absorption of water from all the tissues. Hence the sinking of the eyes, the pinching of the features, the corrugation of the skin of the fingers. This draining off of the watery part of the blood, if not the sole, is an important factor in the arrest of the urinary secretion. The blood does not contain the material for the secretion of urine, but as contributary cause of suppression we must reckon the diminished pressure of the blood in the kidney, from the enfeebled action of the heart. But this cardiac failure is itself, again, partly the result of the thickening of the blood caused by the diarrhœa. The urine is not the only secretion arrested. The secretion of tears, sweat, saliva, and, in most cases, of milk is arrested. We thus see that on the diarrhœa many of the other symptoms of cholera depend. As a rule, when attempts have been made to remedy the evils caused by the loss of fluids, by means of injection of saline solutions into the veins, the rapid improvement of the patient's condition has shown how essentially dependent many of the prominent symptoms of the disease are on the dehydration of the blood; but the fluids injected in this way are rapidly carried off again by the bowel, and the patient is within a short time in the condition in which he was before the fluid was injected.

Vomit.—The vomit of cholera is pale or whey-like, alkaline from carbonate of ammonia, and seldom, and only accidentally, contains the comma bacillus. Among the toxic substances isolated from cultivations of the cholera bacillus no emetic principle has been discovered, so far as we know. Yet such a principle is probably present.

Temperature and Circulation.—The algidity of cholera is to be ascribed to the depressed action of the heart, and also to the thickening of the blood impeding the circulation, especially in the extremities. The depressed action of the heart itself is not the result of a single cause. Brieger has isolated a toxic product of the cholera bacillus which has the effect of lowering the temperature and depressing the heart's action, but it is impossible to doubt that the action of this toxin in producing cardiac depression and lowering of temperature must be increased by the thickened condition of the blood.

Another cholera toxin has been isolated which causes cramps and muscular tremors. We are therefore justified in the present state of knowledge in looking upon the muscular cramps as wholly or in part the result of the cholera toxin.

There is a still more important phenomenon for which we have no satisfactory explanation, namely, the increased temperature of the interior of the abdominal cavity. This appears to be a pretty constant concomitant of algidity. The higher the rectal temperature, the greater the danger. On the approach of death the temperature of the whole body has been found to rise, and it generally continues to rise after death.

A fall of the internal temperature is a favourable sign, and is usually followed by an increase of the surface heat. When reaction is favourable the temperature in the axilla remains normal, or moderately elevated. In the typhoid stage it rises above the normal.

Respiration.—The carbonic acid in the expired air is reduced from the impeded circulation in the capillaries of the lung.

The Urine.—The causes of the suppression of urine in cholera have been already considered.

PROPHYLAXIS

Knowing the sources of the virus, the media in which it grows, the modes in which it is transported from place to place, and the vehicles by which it is conveyed into the body, prophylactic measures can now be applied with a precision and success that were formerly impossible.

The poison ought to be attacked at its source by the cremation or thorough disinfection of cholera evacuations. In cholera hospitals it will be better to cremate the stools after mixing them with sawdust, but their quantity undoubtedly renders this method of disposing of them difficult in private houses. Carbolic acid in 5 per cent solution is the most useful disinfectant for general use. Bichloride of mercury solution, 1 per 1000, with addition of free hydrochloric acid to prevent the formation of albuminate of mercury, is an effective germicide, but its poisonous properties are an objection to its general use. Chloride of lime, 10 parts in solution to 100 of fæces, may also be used. The disinfected stools should be buried at a distance from wells. Clothes may be disinfected by boiling, but as a preliminary they should be fumigated with sulphurous acid. Or disinfection by dry heat may be used. Soiled linen and other articles of little value should be burned. Furniture and utensils should be cleansed by washing with carbolic acid, 1 in 20, added to hot water; they should then be exposed to the sun, so as to be thoroughly dried. Rooms and cabins of ships may be disinfected by sulphurous acid or chlorine. For every 1000 cubic feet 1 lb. of sulphur is to be used. Chlorine may be evolved by adding hydrochloric acid to chloride of lime in the proportion of 22 lbs. of acid to 15 lbs. of the chloride for every 1000 cubic feet of air space. For disinfection of a ship's bilge a 5 per cent solution of carbonic acid, left to act for forty-eight hours, may be employed. We must also mention that Hankin has great faith in permanganate of potash as a means of disinfecting cholera wells. The permanganate is added at sunset, so as to allow the sediment to settle to the bottom. In the morning the water is fit to drink. To our way of thinking, cholera wells should be shut up whenever practicable, and if this is impossible, the water should be boiled before being used for any purpose whatsover.

The measures demanded for preventing the transport of the disease are the regulation of pilgrimages in India, Mecca, and Mesopotamia. Effective sanitation of pilgrim resorts, means for isolating the sick, arrangements for the inspection of pilgrim ships, their quarantine when necessary, and the disinfection of the clothing and other effects belonging to the infected bands, come under this head.

These are matters of international policy, on which it is unnecessary to dwell. Although strict quarantine is impracticable in a country like England, it is of great value in preventing the introduction of the disease in islands and countries where trade is not extensive and is limited to a few ports when properly and humanely carried out.

The measures for removing conditions favouring the growth of the germ—what I have spoken of as breeding-places or culture media—resolve themselves into general sanitation. Moist land should be drained, the pollution of the soil by excreta, and the contamination of the water of harbours and streams by cholera evacuations, and the discharge of sewage prevented. Cesspools are to be disinfected and closed, and all refuse and organic matters regularly removed. Gutters and sewers are to be kept in good order. Streets, yards, and houses should be inspected regularly, and measures taken to keep them in a state of perfect cleanliness. If such means be adopted, the germ will find no medium on which to grow, and even should the disease be imported, it is little likely to spread.

Above all, care must be taken to prevent the pollution of drinking water, which is the vehicle for the wholesale diffusion of the virus. No city dependent on shallow wells or on sewage-polluted rivers is safe from cholera outbreaks. An efficient system of filtration (such as that supplied by some of the London Companies) does, however, materially diminish the risk attending the use of sewage-polluted water. When a water-supply has become polluted, the obvious precaution is to boil it before use.

When cholera breaks out in barracks, the troops should be marched out to a dry and elevated locality having a pure water-supply. If the infection persists, the camping ground should be changed. The old rule of marching at right angles to the wind may be safely ignored. The barracks should, in the meantime, be disinfected and the water-supply looked into.

Cholera outbreaks on crowded vessels are always difficult to deal with, and not easy to prevent. Careful daily inspection, the isolation of the first cases, and attention to cleanliness, and, above all, free ventilation are the most likely means to prevent the spread of the malady. If the weather be fine the patients should be put into boats, hung from the davits and fitted up with awnings, and the utmost attention paid to the disinfection of the berths, cabins, and the belongings of the sick.

One other means of prophylaxis which affords a reasonable hope of immunity is the method of vaccination practised by Haffkine. Of 5357 labourers employed in the Cachar tea-gardens, 2381 were inoculated, while 2976 remained unprotected; 4 of the inoculated and 60 of the uninoculated died. In other words, the deaths were proportionately twelve times more numerous among the unprotected.

Unfortunately the protection does not appear to last above a year or a year and a half, yet the value of this method in the case of bodies of men having to live for a limited time in an area where cholera is endemic, or in the case of a community exposed to an epidemy, cannot be overestimated. If further experience should confirm the reports of the results obtained from these vaccinations, the terrors of cholera will be greatly diminished.

Diagnosis.—With the aid of a bacteriological examination of the stools, there ought now to be little difficulty in distinguishing severe cases of simple diarrhœa from cholera. A well-marked case of cholera cannot be mistaken for any other disease, except, perhaps, the choleraic form of malarial attack; but attention to the history of the case, combined with a microscopic examination of the blood and fæces, will clear up the diagnosis. Poisoning by arsenic, antimony, and corrosive sublimate present many of the features of cholera, but the cholera stools are characteristic.

PROGNOSIS.—We need not say that in a disease, which on an average carries one-half of those attacked, the prognosis should always be guarded. The disease is most fatal in the extremes of life and in pregnant women. Great prostration, well-marked cyanosis, involuntary discharges, or cessation of the diarrhœa without any sign of reaction, and the occurrence of stupor and persistent anuria after reaction has set in are all of evil augury. On the other hand, a fall of the rectal temperature, an increased fulness of the pulse in the algid state, a normal temperature and absence of typhoid symptoms after reaction has set in, and a urine abundant in quantity and free from albumen are hopeful signs.

TREATMENT.—Although the indications for treatment are now better understood, we have as yet no means of successfully meeting those indications. In respect to cholera the advice of Hippocides should never be forgotten, "to do good and do no harm."

The casual indications are to destroy the microbe in the intestinal canal or inhibit its growth.

The *symptomatic indications* are to arrest the diarrhœa and vomiting, to restore the fluidity of the blood, to sustain the heart's action, to restore warmth to the external surface of the body, and alleviate the intense suffering caused by the cramps.

In attempting to fulfil the causal indication, cresline, creosote, cresole, and salol were tried in Hamburg, but none of them proved of any advantage. Calomel did, however, seem to do good, and the benefit resulting from its use may be ascribed to its germicidal properties. One or two 5-grain doses may be given to begin with, and then 3 grains every six hours. Under the treatment the diarrhœa often augments for several hours and then diminishes. Besides calomel there is no internal remedy that can be safely employed to meet the causal indication, and it must be confessed that calomel does so imperfectly.

Considerable success, however, appears to have followed the use of warm tannic acid enemata recommended by Cantani and others, and, if we mistake not, they were found to be of some service in Hamburg when given in the first stage of the disease. The tannic acid is supposed to destroy the cholera bacillus and neutralise its toxins, and it may do so, but the question is, can tannin enemata reach the seat of the disease? Cantani's formula is: Tannic acid, grammes, 3 to 6; gum-arabic, grammes, 30 to 50; laudanum, 30 to 50 drops; infusion of camomile, at 30° C., 1 litre.

The Diarrhœa.—There is something approaching unanimity as to the advantage of arresting the premonitory diarrhœa. It is believed that the disease may frequently be cut short in this stage by the use of astringents and sedatives, and their employment is also indicated in the first stage of the developed disease. When algidity has set in the diarrhœa usually abates, at any rate absorption is then so completely arrested that this class of remedies would then be useless. Compound chalk powder, opium in combination with sulphuric acid, or with acetate of lead, are the remedies most frequently resorted to in order to check the diarrhœa. Graves considered acetate of lead and opium to be the best preparation. He recommended twenty grains of acetate of lead and one grain of opium to be divided in twelve pills, one to be given every half hour until the discharges began to diminish, and he says that "in all cases where medicine promised any chance of relief, this remedy was attended with the very best effects." Perhaps no more reliable remedy to check the diarrhœa can be named than acetate of lead in combination with opium.

Vomiting.—For the arrest of vomiting ice in morsels, chloroform sinapisms over the stomach, calomel in large doses—say, 15 to 20 grains, mustard emetics, and many other remedies have been tried. None appear to succeed better, or so well, as a tablespoonful of mustard in a pint of warm water. Sometimes a massive dose of calomel will quieten the stomach when other means have failed. In the less severe cases, ice, and a large mustard poultice over the stomach, will prove of service.

Collapse.—We have seen that the draining off of the water from the blood contributes to the arrest of the secretions, to the enfeeblement of the heart, and the consequent algidity. The injection of saline fluids into the veins has often been followed by a temporary improvement in all the symptoms; but the injected fluid is rapidly discharged by the bowel, and the patient falls back into the hopeless state of collapse from which the injection had for the time rescued him. Some recoveries have followed this plan of treatment, but its employment has generally ended in disappointment, and it is not even devoid of danger. A safer, but generally an equally unsuccessful, method of supplying the loss of water to the blood is that of injecting water into the subcutaneous tissue. A formula which may be used, if it should be thought proper to try this remedy, is: Water 1000; chloride of sodium, 4; carbonate of sodium, 3. The fluid itself and the instruments must be sterilised. If this is done no harm will ensue, and in a desperate case this remedy might be tried.

With the object of restoring warmth in the state of collapse much benefit will often be obtained from placing the patient in a warm bath. Begbie found it particularly useful in the early stage of algidity when the patient was young and could be easily moved about. During the epidemy in Naples in 1884, Rigoletti employed the warm bath largely in the algid stage with the best results. The bath was prepared with water at the temperature at 100° to 104° F., to which some liquor ammoniæ was added. Under this treatment the diarrhœa continued, but the vomiting and hiccup ceased, the pulse improved, and the secretion of urine was re-established. This method was also found of service in the recent epidemy at Hamburg, and ought to be tried. In milder cases, hot, moist linseed poultices, sufficiently large to cover the whole abdomen, sides, and lower part of chest, and changed *before* they begin to get cold, or the hot pack, along with friction to the extremities, should be resorted to. If stimulants are to be employed in this stage they should be used in great moderation.

Muscular Cramps.—For relief of muscular cramps friction with some stimulating liniment should be employed. If this does not succeed, a tourniquet placed over the upper part of the limb, so as to constrict it, may be tried. The compression should not, however, be continued too long, but having been intermitted, it may if necessary be resorted to again.

To sum up: Our efforts must be directed to stop the diarrhœa in the early stage by astringents. If this fail, or if the patient comes under treatment, after the disease is developed, calomel by the mouth and tannic acid enemata should be employed. In the state of collapse, the warm bath, hot packs, or, in mild cases, large linseed poultices, with friction to the extremities, and a judicious use of stimulants by the mouth or subcutaneously, are the most hopeful means of relief. The typhoid symptoms which often follow cholera are to be treated on the ordinary principles applicable to the special conditions present.

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Cholera Infantum. *See* Gastro-Intestinal Disorders.

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Synonyms.—*Choleraic Diarrhœa, Cholera Morbus, Cholerine, Sporadic Cholera; Diarrhée épidémique* (Fr.); *Sporadische Cholera* (Germ.).

Definition.—An acute, intense inflammation of the gastro-intestinal tract, especially of its mucous membrane, accompanied by copious watery discharges from the bowel, and in most instances introduced by acts of vomiting; simulating in the later stages Asiatic cholera, but giving no evidence of the presence of the "comma" bacillus.

Etiology.—The presence of poisonous bodies, animate or inanimate, within the lumen of the alimentary canal supplies the causative agent of choleraic diarrhœa, the resulting effects varying with the precise nature of the poison and the coincident environment, both locally and generally. Many agents produce symptoms of diarrhœa physically indistinguishable from one another. The stools in true cholera, occasionally in enteric fever and malarial attacks, and those following arsenical poisoning and sunstroke, apart from any specific micro-organisms in them, are very similar in character to the alvine evacuations of cholera nostras.

In each case the presence of the exciting cause leads to inflammation of one, or all, of the coats surrounding the intestine, and frequently of the stomach also.

In choleraic diarrhœa the cause may be organismal, or be derived from the products of organismal metabolism.

Choleraic diarrhœa is, in fact, an inflammation of the digestive canal caused by various agents, but in its results simulating Asiatic cholera more or less closely; absence of the specific "comma" spirillum of the latter disease, and of the symptoms significant of this organism's more intimate and potent influence upon the tissues and processes of the body, serving to distinguish it.

The question of greatest interest for us is concerned with the behaviour and conduct of the bacterial species which normally inhabit the intestinal canal. When of good conduct they serve to control the fermentative processes, brought into being by their own actions, but regulated by reason of the antagonistic properties severally displayed. Commonly peaceable and useful, may they not, stimulated by fortuitous surroundings, acquire and exhibit on occasion a power to do evil? Encouraged by a novel environment, affording them abnormal but more easily assimilable food-stuffs, may they not wax more rapidly in number, and elaborate more poisonous toxins? Appendicitis would appear often to be due to altered characteristics of this kind endowing micro-organisms in the bowel with harmful tendencies. Intestinal dyspepsia and fermentation, enteritis (but a short step further forward), acute gastro-enteric inflammation can be initiated by similar agents in different stages of virulence. The bacillus coli communis, for example, is a normal peaceful inhabitant of the bowel. Usually assisting in the struggle against saprophytic organisms and to check putrefaction, but easily induced, as it seems, to assume new roles, and, as circumstances favour, to become the active exciting cause of inflammatory conditions both in the bowel itself and in the body tissues.

Bacteria of this class may be supposed not only to increase in vigour and numbers more markedly in the bowel under favourable conditions, or to elaborate more toxic poisons, but to behave in the same manner also if present in food-stuffs before ingestion, and these latter, on gaining the bowel, may be able to work evil.

Whenever gastro-intestinal catarrh occurs, some predisposing cause may be predicated with surety. The exciting cause may come from without, or may be generated within; in either case contributory conditions have nearly always preceded. Insufficient food, or food of unsuitable characters, lack of fresh air, weakened physical and mental powers, exposure to extremes of temperature, particularly if indoors, with overcrowded and non-ventilated rooms, render the chance of an attack more probable and more severe.

The most potent contributory cause of attacks of choleraic diarrhœa in babes and sucklings arises from the well-nigh ineradicable belief implanted in the popular mind, that the young were never intended to breathe the fresh air of heaven except during the comparatively brief periods when they are "taken out." Unclean bottles and cups, impure milk, foods unsuited for them at their age, would often prove innocuous, for the body in healthy circumstances is capable of much, if only the child were allowed enough oxygen. Of course, too little food is also a common predisposing cause, and when conjoined to close apartments almost certainly fails to permit of a successful struggle against poisons ingested. No wonder that in enfeebled, marasmic children, hot-house bred, but with less fresh air than is supplied to hot-house plants, starved of oxygen, the bacteria find a congenial soil in which to increase and multiply; little hindrance here to their running *amok*. Milk that is tainted, so common during summer, especially if the supply is delivered but once in the day, too large a quantity of it given at one time, allowing many of the bacteria in it to escape the antiseptic action of the stomach's secretion, a dietary composed of "bites of whatever is going," want of regulation of the action of the bowels, constipation relieved at intervals by strong purgatives,—all are active or accessory causes in infants.

It is probable, also, that poisons absorbed into the circulatory fluids of the body, especially those manufactured by abnormal intestinal processes, when in larger amount than can be dealt with by the liver, in virtue of its protective properties, may occasion an attack of choleraic diarrhœa by a general in addition to a more local action exercised on the nerve endings and fibrils in the wall of the gut during their transit from bowel to vessel.

The actual and true cause of choleraic diarrhœa is almost always to be found in the presence of poisonous products of the metabolism of living forms, and often of the living forms themselves capable of elaborating them. Under ordinary circumstances many of the active agents and some of the poisonous products are unable, by reason of a less activity, a more rapid and thorough neutralisation, to do much harm; but at other times they may be so generated, nourished, manured, and environed as to possess extraordinary powers of development and increase. Instances of choleraic diarrhœa caused by mineral poisons, and many others of the more common chemical substances, fall to be discussed more properly under "Food Poisoning," "Toxicology."

The victims of choleraic diarrhœa, if adults, suffer, as a rule, because of their own indiscretion; from the folly of those put over them, if in childhood.

The attacks of the disease are supposed to be more apt to occur under meteorological conditions favouring heat by day with disproportionate cold by night, or when the air is warm and moist. But these conditions can have but little effect on the mode or season of attack unless accompanying ingress of some poisonous agent or substance. And even then those who avoid the inhalation of re-exhaled, impoverished air, nor sleep in the same, long fasts, sluggish action of the bowel, and lack of muscular exertion, seldom are unable to overcome and inhibit the agents introduced into their bowel (unless if in most unusual quantities), which in those who live conversely would be almost certain to find a fertile soil unprotected and uncared for. Impure water, tainted meat, animal ptomaines are more commonly met with in hot weather; in fact, all bacterial life is more active.

SYMPTOMS.—*In Infants.*—The little victim to one of these attacks is in all probability apparently in a normal state of health up to the actual onset of the symptoms. Of a sudden the contents of the stomach are expelled, generally soon after a meal, in an undigested state, and almost at the same time a more or less fluid discharge takes place from the bowel. The discharge is acid, often green or greenish-yellow in colour, and containing lumps of undigested food. In the majority of cases the vomiting soon ceases, but sometimes the stomach may continue irritable throughout. There may be as many as from twenty to thirty motions during the first twenty-four hours, the later discharges becoming colourless, almost odourless, and in fact similar to those of true cholera. The child suffers from thirst, frequently most intense; from abdominal pain, as the drawing of the legs up against the abdomen and crying show; from local pain round the anus, irritated by the acrid stools; he becomes prostrated, wizened in look; the body temperature falls below normal, the pulse but flickers; and if the diarrhœa persists, muscular cramps, followed soon by general convulsions, set in; death closing the scene. In severe cases a previously strong well-nourished child will lose within but a few hours all his healthy curves and plumpness, to show in his appearance all the signs of extreme marasmus.

At any stage of the attack, however, the symptoms may halt and shortly recede; in even seemingly hopeless cases recovery may result (see "Gastro-Intestinal Disorders").

Adults.—Few premonitory symptoms assert themselves. Severe purging, with or without precedent emesis or gastric nausea, but always accompanied with paroxysmal abdominal pains, frequently of a most agonising character; the passage first of liquid fæces, dark sometimes in colour, light hued as a rule, irritating and paining the anal surface, of more or less foul odour, becoming more and more liquid, with less and less smell and colour, more frequent and copious, in time indistinguishable from the rice-water stools of true cholera, lead to the sharpened features, the cold, clammy goose-skin, the intense thirst, the lessened urine, the cramps and prostration significant of excessive abstraction of fluid and serum from the organism. The prostration, the cold skin, and the actual lowering of body-temperature which may occur are undoubtedly as often caused by reflex action through the nervous centres from an irritation of the local terminations of intestinal nerves as through actual loss of fluid, while as the resultant effect is to withdraw blood from the skin to supply the inflamed intestinal tissues, the discharge of fluid into the lumen of the gut is facilitated.

Should the attack proceed to a fatal ending, the skin becomes cold and livid, the face more pinched, the muscular spasms more general, although towards the end they are replaced by a comatose, quiet state; or again, the mind may almost to the last retain its full consciousness. During an attack the urine is always scanty; in fatal cases it may be completely suppressed for some time before death.

If the attack arise from ingestion of preformed ptomaines, violently poisonous and irritative in character, the time which elapses between their ingestion and the onset of the symptoms varies directly with the character of the meal taken which included them, its amount, and the strength of the gastric juice encountered. If the stomach be empty, or nearly so, and the poisonous agent taken in a liquid, the symptoms occur rapidly; if in solid form, a longer time elapses; while if taken along with all the components of a full meal, the onset of diarrhœa may be delayed until eighteen or twenty-four hours have passed.

If the cause be due to organised forms, the appearance of symptoms is usually longer delayed, varying directly with the virulence of the bacterial type and the facilities afforded it for its growth.

One form of acute diarrhœa, in many instances reaching choleraic intensity, is of interest, although of doubtful etiology. It is every now and then met with in large institutions, especially in the writer's experience in boarding schools. From no discoverable cause a number, perhaps even the majority, of the inmates will be attacked almost simultaneously with acute diarrhœa. Seldom serious, much the larger proportion of those attacked recover speedily; a minority, however, may suffer severely. From what can such an attack arise? Not from food, for all eat the same, and all do not suffer.

PATHOLOGY.—The pathological conditions are akin to those of acute gastro-enteritis. The mucous lining of the gastro-intestinal wall is acutely inflamed over a more or less extensive area. If the irritant cause is very powerful, the inflammatory changes may spread to the other coats of the bowel wall.

Investigation of the watery stools shows them to be almost entirely composed of pure serum, drained by the intestinal glands from the blood. From them various forms of bacteria can be grown, and ptomaines may be isolated by appropriate methods. The bacillus coli communis is constantly, well-nigh invariably, to be found.

DIAGNOSIS.—The most important point in the diagnosis of choleraic diarrhœa lies in its differentiation from Asiatic cholera. This point settled, it is of little moment to be able to distinguish choleraic diarrhœa due to one special form of poison from that caused by another; the symptoms are practically identical, the treatment the same.

A history of previous contact with infection, early onset of collapse, of loss of voice and enfeebled pulse, and the detection of the comma bacillus in the stools suffice to indicate the presence of true cholera.

Occasionally purely nervous diarrhœic attacks closely simulate choleraic diarrhœa; but here a history of former illnesses of the same type, of some nerve disturbance, the comparatively slight discomfort or pain experienced, the character of the dejecta, less offensive and maybe watery from the commencement, and the slower onset of signs of exhaustion, serve to point a difference.

The occurrence of choleraic diarrhœa during the progress of enteric fever is infrequent, and can scarcely be mistaken for other than a complication of the primary disease, except when accompanying an ambulatory unsuspected case.

The liquid stools common in intestinal fermentative dyspepsia chiefly call for evacuation in the morning hours, are intensely fœtid and fæculent, and accompanied by much flatulence.

The symptoms attendant on colitis, mucous and membranous, and the distinctive characters of the motions in these conditions, as well as the differential signs of dysentery and dysenteric diarrhœa, are treated of under their several headings.

PROGNOSIS.—Unless among infants, old people with weakened power of resistance or with feeble hearts, or in chronic invalids of little vitality, choleraic diarrhœa, when independent of ptomaine poisoning, is seldom fatal, although seeming on many occasions to nearly forebode death. In infants fed improperly, housed in badly ventilated or unventilated rooms, it proves very fatal. In adults, when uncomplicated, and where proper chances for treatment are obtainable, it rarely causes death, unless it be the result of poisoning by ptomaines taken with the food in sufficient quantity to overpower all means of successful resistance or treatment.

TREATMENT.—Starvation is the best line of treatment in both infantile and adult choleraic diarrhœa; cold or iced water, and in adults, aerated water, if desired; if well tolerated, a little milk and lime water may be given; rest the affected parts. In such cases one or even two days may easily pass without nourishment being taken. In infants, unless the diarrhœa ceases early, this should be a constant rule; ignore the protests of parents or relatives. In adults when a desire for food returns, apart from the simple craving for fluids, milk diet may be safely begun.

In adults the less medicine given the better. Cold or iced water, hot-water bottles to the feet and legs, hot poultices, with or without mustard, over the abdomen, and plenty of fresh air generally suffice to arrest the symptoms. The administration of opium or morphina is of questionable propriety. The poison in the alimentary tract merits removal rather than detention; but if the pain be great, and the reflex nervous prostration pronounced, free use of opium will help to remove any chance of immediate collapse. If the patient be in a less prostrate state, the application of warmth, a small dose of calomel,—large doses of calomel are dangerous, while small amounts are better fitted to act antiseptically,—and half-ounce or even ounce doses of whisky or brandy, often arrest the symptoms. If painful cramps supervene, etherisation is advisable, indeed the calmative effect of an anæsthetic upon the whole nervous system, and the relief obtained from the pain of cramp and from the local abdominal pain, suggest that an earlier employment of an anæsthetic in such cases might prove of great service. If seen early, administration of castor oil and laudanum together act well.

Should the stomach be restive and reject the stimulants ordered, or the patient continue to retch, dilute hydrocyanic acid may be given, or teaspoonfuls of brandy and water, in which one drachm of the spirit is added to two or three ounces of water, the dose to be repeated every few minutes if vomiting threaten. Should the diarrhœa be unchecked by such measures, the sedative powers of opium or morphina may be called upon. The most efficacious method undoubtedly lies in the use of morphina suppositories.

A day or two after the active symptoms have abated, especially if they have been arrested by the use of opium preparations, a full dose of castor oil may be given with safety to clear out all that remains in the bowel.

The general indications for treatment may be summed up in a few words. If the patient appears to be strong enough to bear it, remove the exciting cause; if collapsed, and in danger from persisting exhausting discharges from the bowel, stimulate, and introduce morphina in a suppository or under the skin; in all cases apply warmth to the abdomen and the lower limbs, stop all food by the mouth,—in adults milk may be allowed,—inculcate rest, and secure the presence of fresh air.

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Chordee. *See* Penis.

Chorea.

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The name "chorea" has unfortunately been applied to a number of entirely different complaints, the common feature in all "choreic" diseases being the occurrence of involuntary, irregular muscular movements.

Common Chorea

(Chorea Minor; Sydenham's Chorea; St. Vitus's Dance) consists in a group of characteristic symptoms of subacute onset and chiefly occurring in children.

ETIOLOGY.—*Age.*—Chorea is essentially a disease of childhood and adolescence, the vast majority of cases occurring between the ages of five and fifteen years. After fifteen years of age chorea is much less common as a primary attack, although second and third attacks are by no means uncommon after puberty. After the age of twenty chorea becomes still less frequent, although no age is exempt and the disease may develop even up to an advanced age, as in one form of *chorea senilis.*

Sex.—The female sex is much more liable to this disease than the male, the average proportion being three girls to one boy. After the age of fifteen there is a still greater preponderance of female patients.

Hereditary Influence.—A family diathesis towards rheumatism is very common, and should be inquired for in every case of chorea. In a considerable proportion of cases a family history of nervous diseases can also be obtained.

Rheumatism and Endocarditis.—Experience shows that a very large number of cases occur in patients who have had articular rheumatism. Many patients develop rheumatism in the course of an attack of chorea or at a subsequent period. Also many cases of chorea are preceded or followed by endocarditis. The endocarditis is sometimes discovered for the first time during the course of the chorea, and it is rare to find a patient with a second or third attack of chorea in whom the cardiac sounds are normal.

In a recent series of forty-one consecutive cases of chorea, in twelve of them (including a primigravida) there was a history of previous rheumatic fever; another patient developed pain and swelling in both wrist joints whilst under observation, within a week after the onset of the chorea; whilst another primigravida, who had previously had two attacks of chorea, developed pain and swelling in one knee joint a fortnight after the commencement of her attack of chorea gravidarum. Of the remaining twenty-seven cases, fifteen had a strong family history of rheumatic fever, whilst in the remaining twelve there was no history of rheumatic fever either in the patient or in the patient's near relations. Yet out of these twelve no fewer than five had mitral regurgitation, one had mitral stenosis which ultimately proved fatal, and only six had no valvular affection of the heart.

Scarlet fever with arthritic manifestations is recorded in a few cases to have been a direct antecedent of chorea.

Fright, emotion, traumatism or some variety of shock to the nervous system is a fairly common antecedent to an attack of chorea, although the majority of cases develop without such an exciting cause. Still a history of some fright or shock is obtained in from twenty to thirty per cent of cases. Mental emotion as an alleged exciting cause is more common in adolescents, from the age of sixteen upwards, than in childhood. But the study of a number of cases of chorea attributed to emotion shows that the vast majority of them occur in constitutions which are already rheumatic. Moreover, the history of mental shock is often an indefinite one, and early choreic symptoms are often present before the advent of the emotion which exaggerated them. In the above series of forty-one cases, fifteen gave a history of some fright or mental shock, but of these fifteen only one was free from evidences of rheumatism, twelve of the others having either had rheumatic fever or mitral disease and the remaining two having a very strong family history of rheumatic fever.

It used to be thought that chorea may spread from one patient to another by *imitation.* Small epidemics of choreiform movements, such as have now and then been recorded in girls' boarding-schools, are really hysterical in nature and not truly choreic. A primarily choreic child, however, may have a relapse of true chorea from the excitement of seeing another patient with a similar affection.

The presence of *worms* in the intestinal canal has been believed by some to cause the disease in certain cases by reflex irritation. But in the above series of cases the only case of chorea in a child with tape-worm occurred in a girl who had previously had an attack of rheumatic fever.

Pregnancy undoubtedly acts as an exciting cause in certain patients, the disease developing in the early months of gestation, usually from the third to the fifth month, either in previously healthy patients or more frequently in cases with a previous history of rheumatism or of actual chorea. Young primigravidæ are chiefly so affected, frequently unmarried girls. The connection between the chorea and the pregnancy is so close a one that when the pregnancy comes to an end, either in a normal manner or by abortion, the chorea ceases. Frequently the disease recurs with succeeding pregnancies (*vide* " Chorea Gravidarum ").

SYMPTOMS.—The disease is gradual in onset, usually taking a day or two to develop sufficiently to call attention to the presence of any abnormality. The patient is often fretful and more irritable than usual; then restless, wriggling movements are observed in the limbs, face, and trunk. The child at school cannot sit still, and the teacher complains that the patient's handwriting is clumsy and untidy. Often the patient drops things suddenly out of the hand. Sometimes this condition is not recognised as being really due to disease, since a degree of motor restlessness is present in many children during mental embarrassment or other emotion. The choreic child is often considered at first to be merely badly behaved, but as the symptoms develop, it becomes evident that there is some underlying diseased condition.

In a typical, fully developed case of chorea the symptoms may be classified into various groups, viz.: (*a*) irregular, involuntary movements; (*b*) inco-ordination on attempted voluntary movements; (*c*) weakness of the affected limbs; (*d*) a variable amount of psychical disturbance.

The irregular, involuntary movements are quite characteristic. They are of a twisting, wriggling, tossing type, quite irregular in time, and constantly varying in character and degree. The patient cannot stand or sit still, but is in a state of continual motor restlessness—" fidgeting." The movements are usually first noticed in the arm or face, the legs, as a rule, being less affected. The shoulder may be suddenly shrugged, the arm abducted or adducted, the elbow flexed or extended, the hand pronated or supinated, the fingers spread out, flexed, or extended, and these sudden irregular movements are often combined in a complex, confused fashion.

Similar movements are also seen in the face. The brow may be suddenly wrinkled, or brief grimaces may be made, the mouth being drawn to one side, the lower lip everted, the mouth pursed up, the upper teeth exposed, or the eyes screwed up. The ocular muscles also participate, the patient does not fix objects steadily, but looks restlessly hither and thither and may now and then squint momentarily. The jaws may open and shut irregularly, the tongue may be rolled about in various directions and not infrequently it is bitten by a sudden involuntary snap of the teeth. Sometimes the soft palate is seen to move irregularly up and down. The movements of the muscles in and around the mouth may produce peculiar sucking, smacking, or slobbering noises.

The trunk is often rotated to one side or the other and the head suddenly turned in various directions. Respiration is frequently jerky and irregular. The action of the diaphragm is often overshadowed by that of the intercostals and of the extra muscles of respiration. Sometimes the diaphragm and intercostals contract alternately instead of synchronously. In a few cases the laryngeal muscles are affected, so that jerky laryngeal noises or groans are produced.

Choreic movements in the legs are usually of a much less complicated type than in the upper extremities. The patient cannot stand placidly, sometimes one foot is moved, sometimes the other. Walking is only impaired in severe cases. The knees may suddenly give way during progression, or the movements of the legs may be so violent that the patient is unable even to stand.

The choreic movements vary in degree in different cases, from the slightest restlessness of the fingers to the wildest and most violent, irregular movements of all the voluntary muscles of the body. The affected joints are impetuously flexed, extended, rotated, or circumducted, and in severe cases the limbs and trunk may be thrown about so violently that the patient may fall out of bed, or may bruise or cut the bony prominences of the head, trunk, or limbs by throwing them against surrounding objects.

The movements usually commence in the hand, spreading later to the face, trunk, and leg. They generally affect one side of the body earlier and more severely than the other. Often they remain confined entirely to one side (*hemichorea*). In a few cases they migrate completely from one side to the other. As a rule, in right-handed patients the left side is more commonly affected than the right; but this is not invariably the case. Sometimes the movements affect both sides with equal intensity.

Voluntary movements are more or less interfered with, when involuntary choreic movements are going on. The patient executes voluntary movements hastily and spasmodically as if attempting to seize an instant when choreic movements are absent. Sometimes, however, voluntary movements are almost steady, whilst involuntary movements are well marked, but more commonly choreic movements are increased by voluntary movement. A good test for slight cases of chorea is to make the patient hold up both hands for a few seconds above his head with the fingers outstretched. This usually succeeds in bringing out a few irregular, wriggling movements of the fingers on the affected side. Or make the choreic patient squeeze one's hand, and the grasp is perceived to be sudden, spasmodic, and ill sustained. If the grasps be compared on the two sides in a case of hemichorea, it will be found that the grasp on the choreic side, although more sudden, is yet actually weaker than on the unaffected side. Sometimes when picking up objects the patient makes a sudden dash for the article and often drops it after securing it. Sometimes, again, the patient has a difficulty in letting go an object when he wishes to do so. Thus in feeding himself he often drops his cup, and scatters his food about the table with his knife and fork.

In the lower limbs, in addition to the involuntary movements already described, the gait may be peculiarly altered. The knees may suddenly give way during walking, causing the patient to fall.

When the tongue is protruded it is shot out suddenly and pulled in again with a jerk. Articulation is often jerky and indistinct. Words are shot out, suddenly cut short, or interrupted by the irregular respiratory movements. In bad cases the speech may be quite unintelligible, or the patient may speak only in whispers, or may even absolutely refuse to speak at all for days or weeks. In the latter instance there is a superadded psychical element present.

Severe affection of the lips, tongue, and pharynx may cause incoordination of swallowing, and the general nutrition may thereby be gravely affected.

The motor unrest is greatly increased by emotional states. The excitement of a medical examination often renders the movements much more violent. Scolding the patient by teachers or parents only makes the child more restless. Sometimes the patient sleeps badly. As a rule, during sleep the choreic movements entirely cease; but in very rare cases exactly the reverse condition is found (*chorea nocturna*), in which the movements are greatest during sleep, disappearing almost completely when the patient is awake.

A considerable degree of muscular weakness is common in chorea, and in some cases this may be so severe that the patient is unable to raise a limb from the bed, although slight restless movements are seen going on irregularly in the weakened limbs. But the patient can always execute voluntary movements, however feebly, at all joints in the apparently paralysed limbs. There is never complete paralysis of any group of muscles. Such cases, where the weakness is out of all proportion to the choreic movements, have been termed "paralytic chorea," and in many of them the choreic movements are only elicited on attempted voluntary movement. In some cases muscular paresis is the earliest symptom of the disease. The patient's relations notice that the child uses one arm less than the other, and finally that he does not use it at all. As the disease recovers the paresis fades, and the choreic movements become more evident.

The muscles remain normal in volume. Their electrical excitability is sometimes increased on the affected side, both to the continuous current and to induction shocks.

Sensory abnormalities are more frequent in chorea than it is generally supposed. It is true that choreic patients do not usually complain of pain, numbness, or any abnormal feeling, but if the cutaneous sensibility be carefully tested in every case, it will be found that a considerable proportion of patients exhibit a slight degree of blunting of sensation on one side of the body. This was so in ten cases out of forty-one in the above series. Such hemi-anæsthesia, when present, is always on that side of the body on which the choreic movements are most marked. Sometimes the visual fields are concentrically contracted, and the acuity of vision, smell, taste, and hearing may be diminished on the preponderatingly choreic side, with or without slight hemi-anæsthesia to touch or pain. Such affection of the special senses is strongly suggestive of a hysterical element superadded to the chorea, but the frequency of hemi-anæsthesia is very striking. The hemi-anæsthesia of chorea is very slight in degree, and can only be detected by careful comparison of corresponding points on opposite sides of the body. When this is tried a considerable proportion of patients will be found to perceive light touches and pricks more acutely on one side than on the other, and the boundary of this area of blunted sensibility will always be found to lie in the middle line of the body. Sometimes the trunk and limbs are alone involved in the hemi-anæsthesia, the face and scalp escaping.

As to the reflexes, the skin-reflexes are occasionally diminished on the hemi-anæsthetic side. The deep reflexes may be normal, but sometimes they are difficult to elicit. In other cases we may find the "choreic knee-jerk." This differs from the healthy knee-jerk in the fact that when the patellar tendon is tapped, instead of the normal brisk contraction of the quadriceps extensor, followed at once by a sudden relaxation, the knee in such choreic cases is suddenly extended to its full extent and remains so for a second or two, the foot and toes meanwhile exhibiting irregular choreic movements. Ordinarily the sphincters are normal, except in very bad cases when extreme mental dulness causes incontinence of urine and fæces.

In chorea the optic discs are normal. In rare instances slight optic neuritis has been observed. This is probably not referable to the chorea itself, but due to some other concomitant cause. The pupils are often dilated, but they react normally. The temperature is normal, except in very severe cases, when it may rise.

In the majority of cases a psychical element is present. This may be trivial in degree, merely amounting to a certain irritability of temper or a tendency to cry or to giggle on slight provocation. Sometimes, however, there is impairment of memory and distinct mental dulness. The mental element may be quite out of proportion to the severity of the other symptoms. Mental affection is more common in adolescents than in young children. It usually takes the form of depression. Hallucinations may develop with delirium, and the patient may pass into a state of violent maniacal excitement. This maniacal form (*chorea insaniens*) reaches its maximum intensity at the acme of the chorea. It may last several weeks, and generally passes ultimately into a condition of mental apathy in which the patient lies in bed like a log, refuses food and passes both urine and fæces into the bed. As the patient slowly recovers, there is often a transient stage of mild delirium with "persistent ideas." Severe mental symptoms are more common in the chorea of pregnancy than in any other variety of chorea. The ultimate prognosis, however, as a rule is good; most cases of choreic insanity recover completely.

The urine in chorea contains an excessive amount of urea, of phosphates, and sometimes also of hæmatoporphyrin.

COMPLICATIONS.—Of these the most important is endocarditis, which occurs so frequently that it may almost be considered as part of the disease. Choreic endocarditis chiefly affects the mitral orifice and is usually of the simple type. The heart frequently recovers completely and its bruits entirely disappear. Sometimes, however, the valves may remain permanently damaged. Ulcerative endocarditis with embolism is rare.

Acute articular rheumatism may precede, accompany, or follow an attack of chorea. When occurring simultaneously with chorea, it is usually mild in type, evidenced chiefly by joint pains and moderate pyrexia, with little or no joint swelling to be made out on objective examination.

Some choreic children have characteristic "subcutaneous rheumatic nodules," varying in size from that of a pea downwards to that of a sago-grain. They are distributed most commonly along the subcutaneous borders of the ulna and tibia, also around the elbow, knee, and ankle joints, and along various tendons around the wrists and ankles. Sometimes these nodules are only discoverable during the attack of chorea. Erythema nodosum is sometimes present. A painful contracture of the palmar fascia may also occur during chorea, clearing up under anti-rheumatic treatment.

Cases of chorea with intense mental symptoms have already been referred to. Such maniacal cases occur chiefly in adults, and especially in the pregnant state.

Epilepsy as a complication is very rare. Hysteria may complicate chorea, and hysterical movements may sometimes simulate chorea; but the practised observer has usually little difficulty in recognising the existence of a hysterical element when present. Hysterical movements are usually somewhat different in type from those of true chorea, they are often sudden and shock-like and tend to be more rhythmical than in true chorea.

Duration.—The duration of an attack of chorea is very variable. The more severe the attack, the longer will be its probable duration. The average duration is from two to three months, but an attack may clear up in two or three weeks, or it may last six months or longer. Only in exceptional cases does chorea persist continuously for a year or more.

As a rule, the older the patient is when first affected by chorea, the longer is the duration of the attack likely to be.

Chorea is a disease which is very prone to recur. One or two relapses are common, and as many as nine relapses have been recorded. The intervals between two successive attacks vary from a few weeks to several years; the average interval is about one year. In some cases the patient has an attack of chorea regularly at a certain season of the year for several successive years; but as a rule the attacks recur at quite irregular intervals. Relapses are often slighter in severity than the primary attack, but the reverse is sometimes the case.

Prognosis.—In most cases recovery is complete, the inco-ordination gradually ceases and at last the involuntary movements fade away. Serious cases are those in which the movements are of extreme violence and in which insomnia, delirium, and rapid emaciation supervene. Those may end fatally from exhaustion. Sometimes death results from rheumatic fever with hyperpyrexia, or from cardiac failure secondary to valvular disease. Injuries to the skin produced by the violence of the choreic movements may result in intractable sores and the patient may die from pyæmic infection.

In the common chorea of childhood the danger to life is but slight, the average mortality being less than three per cent. Most of the fatal cases are first attacks; death from a recurrence is rare. Chorea occurring for the first time at or after puberty is much more dangerous, since severe heart-lesions are more frequent than in the chorea of childhood.

Chorea in pregnancy is still more serious, nearly twenty-five per cent of cases proving fatal. In such cases the choreic movements are usually very violent, mental complications are common and severe endocarditis is relatively frequent. Many cases abort, and in the patient's cachectic state abortion or even normal labour is dangerous.

In extremely rare cases juvenile chorea may persist as a permanent chronic disease, with or without slight remissions, in spite of all treatment. This form of chorea is commoner in males than in females, and seems to be less definitely associated with rheumatism and endocarditis than ordinary chorea is (*vide infra*, " Chronic Progressive Chorea ").

Chorea developing after middle life is much more likely to be a permanent disease than the juvenile form.

Pathology.—Chorea has a clinical rather than a pathological existence, and the disease is at present classed amongst "functional" disorders. Practically nothing is known of its essential pathology. It is only in severe and complicated cases that the disease is fatal and at post-mortem examinations very varied and inconstant appearances have been found, amongst which may be mentioned general hyperæmia of the brain, embolism or thrombosis of cerebral vessels, and minute perivascular hæmorrhages in the brain and spinal cord. Sometimes fine changes in the nerve-cells of the central ganglia have also been described. But an insurmountable objection to the acceptance of any one of the above as the essential lesion of the disease lies in the fact that any or all of these changes may be absent and the results of examination may be negative, the brain, spinal cord, and peripheral nerves appearing absolutely normal—so far, at least, as the present methods of neuro-pathological research enable us to judge. Recent endocarditis, however, is found in a great majority of fatal cases of chorea.

Some time ago the disease was supposed by some to be due to extensive capillary embolism, and this view was thought to be strengthened by the fact that the injection of starch granules into the carotid arteries in dogs produces choreoid movements. But most cases of fatal chorea show no signs whatever of such embolism. Moreover, "canine chorea" is essentially a different disease from chorea in the human subject.

The nature of the choreic movements, together with their cessation during sleep, points strongly to their origin from some irritable condition of the motor cells of the cerebral cortex. Such a hypothesis is further strengthened by the frequency with which the symptoms are confined to one side and by the frequent presence of mental disturbance.

For want of a better explanation chorea is at present classified as a "functional" disorder of the nerve-cells. But there can be little doubt that some molecular or chemical change in the neuron underlies all so-called "functional" disorders, though as yet we are ignorant of the precise nature of such changes.

It is not at all improbable that chorea in many cases may be due to a toxin, probably closely associated with the rheumatic poison, if not identical with it.

That emotion frequently plays a part as an exciting cause is not antagonistic to such a theory, for the mental shock in such cases often merely precipitates an attack in an unstable or imperfectly developed nervous system already partially poisoned by a toxin in the blood. Endocarditis is as frequent in cases which have followed upon mental emotion as in those which have arisen spontaneously, and yet no one doubts that endocarditis is due to the existence of a poison in the blood. Endocarditis is so common in chorea that we conclude there must be some close connection between the two, and the fact that chorea so often precedes the endocarditis shows that the latter cannot be the cause of the chorea. We are therefore again driven to the conclusion that some common cause must bring about both the chorea and the endocarditis.

DIAGNOSIS.—Ordinarily chorea can be diagnosed at a glance. The peculiar movements are quite characteristic, and the history of a recent onset distinguishes them at once from athetosis following cerebral palsy in early life, which is the commonest condition in which similar movements occur. The movements of athetosis have unfortunately been termed "post-hemiplegic chorea," but the diagnosis between the two conditions is usually easily made, not only from the "athetoid" character of the movements, but also from the presence of muscular rigidity in the athetotic limbs, with a history of some previous cerebral attack (*vide* "Athetosis," vol. i. p. 399).

The rare condition called General Convulsive Tic ("Tic Générale") simulates chorea even more closely, but can be differentiated by observing the systematic nature of the movements in Convulsive Tic, the relatively long intervals of rest between the movements, the fact that the patient can always intermit them in order to execute voluntary actions, and the frequent presence of echolalia, coprolalia, or other explosive utterances and of forced movements.

Sometimes difficulty arises in the diagnosis of cases of "paralytic" chorea, where the outstanding symptom is loss of power in one arm. When a child between seven and twelve years of age is said to have gradually lost the use of one arm, without paralysis of leg or face, the disease, as a rule, is chorea. Careful observation in such cases never fails to reveal slight choreic movements in the affected limb.

The few cases of chorea where the legs are chiefly affected may simulate ataxia of gait, but the choreic movements of the limbs in the sitting and recumbent posture are sufficient to differentiate the two conditions.

TREATMENT.—The mental and motor symptoms of chorea, together with the marked influence which emotion and exertion, mental or physical, have in exaggerating the choreic movements, make it obvious that a most important element in the treatment of chorea consists in the removal of all causes of emotional excitement and physical or mental fatigue.

The patient must not go to school. Lessons must be stopped at once and rest in bed should be insisted on, for several weeks at least, even in mild cases. When the patient is afterwards allowed up, this should only be for a short time each day, gradually lengthening the time spent out of bed.

All sources of mental irritation must be avoided, and only such occupations should be permitted which interest the patient without fatigue or excitement. The child must not be frightened or threatened. A large airy room, if available, should be chosen for a bedroom and cheerful companionship should be provided, such as that of the mother or of a judicious nurse, since mental depression might undo the good produced by rest. If there are other children in the house, they should not be allowed to go into the patient's room, lest they increase his mental and physical unrest.

In cases with violent movements, care must be taken to prevent the patient from falling out of bed or injuring himself. This can be accomplished by laying the patient's mattress on the floor in a corner of the room, well padded with cushions or pillows, to prevent him from knocking himself against the wall. If bed-sores threaten, a water-bed should at once be procured.

Treatment in an asylum is seldom necessary, except in cases with severe mental symptoms where isolation at home is unattainable. But if a case is doing badly at home, transference to hospital is often advantageous and expedites the cure.

The diet should be nutritious and easily absorbed. The more food the patient can take, the better. In severe cases where deglutition is impaired, or where from mental dulness the patient does not take enough nourishment, nasal feeding must be resorted to. Hughlings Jackson gives alcohol —a teaspoonful of brandy every four hours—in severe cases of chorea, and with excellent results. The bowels must be kept open, but strong cathartics are to be avoided.

It is important that the patient should sleep well, and hypnotics must be given if necessary.

As to drugs, arsenic is the classical remedy. It is given by the mouth, and the dose should commence with three or four minims of the liquor arsenicalis three times a day, increasing gradually to ten or fifteen minims. If symptoms of arsenical poisoning appear, such as conjunctivitis, nausea, and gastric pain, the drug must be stopped for a few days and then resumed in a somewhat smaller dose. The administration of large doses of liquor arsenicalis (fifteen to twenty minims three times a day) from the first, for a period not longer than a week, has sometimes yielded good results; but if this mode of administration be persisted in for a long period, it is not devoid of risk. Arsenic in large doses administered for a lengthened period not only induces a brownish pigmentation of the skin but has sometimes produced very intractable peripheral neuritis.

In severe cases chloral hydrate is of great service as a hypnotic and general nervous sedative. Some physicians keep the patient continuously asleep for weeks by means of this drug, but caution is necessary in such cases, since the chloral sleep is sometimes followed by maniacal excitement.

Bromides are of singularly little value in chorea. In very violent cases morphia may be administered hypodermically, or it may even be necessary to give chloroform to control the violence of the movements, but such treatment can only be of temporary service, it being much better to keep the patient under the influence of chloral. In some cases antipyrin is useful but care must be taken not to produce cardiac failure. Therefore, during treatment by antipyrin, the patient must be kept absolutely at rest.

Valerianate of zinc, oxide of zinc, hyoscine, cannabis indica and physostigmine have all been employed, but are of little use. Salicyl compounds occasionally give good results, especially salol; but in other cases, again, they fail entirely.

The application to the spine of electricity, faradic or galvanic, and the freezing of the skin over the spine by means of ether spray, which have sometimes been employed, are of doubtful value; and when we remember that the disease is of cerebral and not of spinal origin, this can readily be understood.

During convalescence mild gymnastics are often of service, and a change of air often serves to confirm the cure.

Finally, it must be remembered that chorea is a disease which is prone to recur. Each successive attack must be treated on the same principles, rest and feeding being more important than medicinal therapeutics.

HEREDITARY ADULT CHOREA (Huntington's Chorea)

This is a somewhat rare disease which must be carefully distinguished from the ordinary chorea minor.

The disease was described in 1872 by Huntington of New York as a form of chronic progressive chorea occurring hereditarily in certain families through many generations, the malady commencing in middle life, being associated with mental weakness and continuing until death. Since its original description the disease has also been observed with considerable frequency in Britain and on the continent of Europe.

The complaint is markedly hereditary in certain families and has been traced through many successive generations. Sometimes, however, one generation escapes and the disease does not afterwards reappear in the family.

Males and females are equally affected, and both sexes may transmit the disease, which begins, as a rule, in middle life, commonly between the ages of thirty and fifty years.

It may arise without any cause whatever, but sometimes it first becomes evident after emotional excitement. Thus in one woman, whose mother and maternal grandmother had been similarly affected, the earliest symptoms came on after the physical and mental shock of being forcibly discharged by her employer, who threw her down a flight of stairs.

Symptoms.—The patient exhibits movements of a choreic type, usually first noticed in the face or upper limbs, gradually increasing in severity and ultimately, in the course of years, affecting all the voluntary muscles of the body. The motor symptoms are similar to those of chorea minor, with grimacing, gesticulation, affection of articulation and almost constant play of involuntary irregular movements of the eyes, face, limbs and trunk. The ocular muscles do not necessarily escape, as some authors state. Sometimes one side of the body is affected for years before the other side is attacked.

The movements, as in chorea minor, cease during sleep and are increased by emotional excitement; but, unlike ordinary chorea, they can be arrested when the patient desires to execute any voluntary movement: thus the patient can still write or thread a needle, even when the disease is far advanced.

Even up to a late stage the patient is able to walk but the gait is often curiously irregular. The trunk and limbs undergo irregular spontaneous movements, the patient often suddenly standing still after walking a few steps.

Sensation is normal. The deep reflexes are generally somewhat increased. The heart is unaffected.

Mental weakness is usually present, commencing as mere "facility" and gradually advancing towards a degree of dementia. The patient is often depressed and suicidal tendencies have sometimes been observed. The patient may at first be irritable or excitable but, as the disease progresses, the mental state usually becomes one of apathy. The mental affection is an insidious one and generally develops some years later than the muscular restlessness, but this is not invariably the case. Mental weakness may precede the motor phenomena.

The disease is an incurable one and its symptoms persist throughout life, but life is not necessarily shortened thereby. The complaint may last from ten to thirty years, and the patient ultimately dies either from exhaustion or from some intercurrent affection.

Diagnosis.—This disease is distinguished from common chorea chiefly by the history of heredity, which in ordinary chorea is very rare; also by the age of onset, the tendency to dementia, the prolonged duration, and the progressive nature of the malady. Chorea senilis, which may either be chorea minor occurring at an advanced age, or may be a variety of chronic progressive chorea, is free from any hereditary tendency and unassociated with progressive dementia.

Pathology.—As in chorea minor, the essential pathology of this disease is quite obscure. It is probably a degenerative process affecting the cortical motor neurons. Very various morbid appearances have been found at autopsies, including slight diffuse chronic meningitis, changes similar to those of general paralysis of the insane, pachymeningitis giahmhoraerca, disseminated miliary nodules of chronic inflammation scattered in the white and gray matter, etc., but these are all probably accidental, and not essential to the disease.

Treatment can only be symptomatic. Arsenic is of little or no use. Tonics and various sedatives may be tried but therapeutic results have, so far, been entirely negative..

CHRONIC PROGRESSIVE CHOREA

Apart from Huntington's chorea, other forms of chronic progressive chorea occur unassociated with any hereditary tendency. Isolated cases may begin in old age, in adult life, and recently have also been observed in adolescence (*developmental chorea*).

These cases are distinguished from ordinary chorea by their duration and by the absence of rheumatic affections or of cardiac complications. The motor symptoms are exactly those of chorea minor, but they become progressively worse and last throughout the patient's life.

Such varieties of chorea are obviously different from ordinary chorea minor which, though it tends to relapse, ultimately, in most cases, becomes cured. They are probably dependent on a chronic degenerative process in the cells of the motor cortex, rather than upon any toxin, as in the case of chorea minor.

ELECTRICAL CHOREA (Dubini's disease)

This is a very rare endemic disease, which occurs chiefly in Northern Italy and Lombardy. It has been observed in both sexes and at all ages. The disease is a progressive one. It commences with a short stage of pains in the head, neck, or spine, soon followed by involuntary spasmodic movements which differ from those of ordinary chorea in being short, sudden and shock-like, similar to those elicited by electrical stimulation.

These contractions usually commence locally in one arm or one side of the face, later, they attack the leg on the same side, and at last they cross over and invade the muscles of the opposite side. In addition, there are sometimes epileptiform convulsions, which may be unilateral.

As the disease progresses, paralytic phenomena are superadded, commencing in those limbs in which the contractions were earliest and most marked. The palsy, which is accompanied by muscular atrophy and diminution of faradic excitability, spreads all over the body and becomes general. There may be considerable elevation of temperature. Throughout the disease the mind is unaffected.

The complaint may last for days, weeks, or months and is usually fatal. Death results from cardiac failure or from coma. Only in a few cases does recovery take place.

The nature of the disease is undetermined. No constant morbid changes have been found. On the whole, it seems probable that the disease is due to some toxin of endemic origin which attacks first the cerebral cortex and secondarily the spinal cord.

Diagnosis.—This disease has not been observed except in Italy. Elsewhere the only complaints which may simulate electrical chorea are myoclonus multiplex (*q.v.* "Muscles") and certain varieties of hysterical spasm, both of which conditions have only to be known in order to be readily distinguished from Dubini's disease.

The so-called *Chorea major* (Chorea magna) must be carefully differentiated from all the truly "choreic" disorders. It is not a chorea at all, but merely a variety of hysteria easily distinguished from true chorea.

CHOREA GRAVIDARUM

Chorea, rare in the adult, is perhaps a little less rare in the pregnant. This is, however, by no means certain. Of 439 cases in the British Medical Association's collective report, 68 were females between 16 and 40, and of these 7 only were pregnant; of 28 delirious female cases, over 15 in the Kommune-hospitalet at Copenhagen, 7 were pregnant and 1 recently delivered. The proportions in these instances are 1 in 9 and 1 in 3, while the proportion of pregnant women to adult women in general is between these ratios and nearer the higher. Nevertheless, the association of chorea and pregnancy is important both for prognosis and treatment.

Cases of chorea in pregnancy exhibit the same relations to fright, rheumatism, and endocarditis, hysteria, family tendencies, and previous chorea, as cases of chorea in general. Fifty-nine per cent of first attacks occur in the first pregnancy, but the ratio of illegitimacy is little if at all in excess of that of women in general. Corresponding to this, the age at the first choreic pregnancy is usually under 25, but cases are on record at all ages up to 40. Chorea may recur in several pregnancies, the largest number of recorded attacks being 5, but choreic pregnancy may be preceded or succeeded by pregnancy free from chorea. Fully half of the attacks began in the first three months, and a third in the middle three months. A few cases are on record where chorea began during the puerperium, and two or three somewhat later.

Of cases where the pregnancy ends spontaneously two-thirds are delivered at term, the proportion of abortion being thus higher than in general. In about 40 per cent of the cases that recover the chorea terminates before the end of the pregnancy. There is no evidence to show what influence the death of the fœtus, before delivery, has on the chorea; in a few cases the perception of the fœtal movements has exaggerated the chorea. Recovery after delivery is, as a rule, gradual, as it is in chorea apart from pregnancy, but in a few recovery is sudden; in one case the chorea became chronic. The mortality cannot be exactly determined, but is certainly less than 1 in 5, and a considerable number of the fatal cases die from causes other than the chorea. In less than 3 per cent of the cases insanity followed the chorea, recovery taking place in a few weeks or months. In a number of cases infective processes have complicated or followed the chorea.

The treatment of chorea in pregnancy is primarily the same as the treatment of chorea generally. Arsenic and salol, and the sedatives and narcotics, including general anæsthesia, must be employed according to the indications of the individual case. Where the case is severe or becomes worse in spite of such treatment, the induction of abortion or of premature labour is justified. In practice this has not always given satisfactory results, but the cases were of great severity, and sometimes the delay had been too great. The induction should be made under anæsthesia, which may reasonably be prolonged till the completion of the delivery. In some cases digital dilatation of the cervix has stopped the chorea. In less severe cases and those which improve under general treatment, it is best to wait spontaneous labour and to watch that, interfering only if this seem specially required.

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Choreic Insanity

There is probably no special form of insanity that can properly be called Choreic Insanity. In every patient suffering from chorea there is usually some mental disturbance. The patient may be dull and listless with apathy and loss of memory; but these mental symptoms being so slight they are overlooked; on the other hand, however, they may become so severe as to call for immediate treatment.

Etiology.—Mental complications with chorea are more common with pregnant women, girls, and young men than with children. Usually there is an unstable inheritance.

Symptoms.—1. *Choreic Mania.*—The excitement rarely begins before the end of the first and after the fourth week of the outbreak of the chorea. Often it is most difficult to say when the bounds of sanity have been passed, as the impulsiveness, agitation, or loss of control grow gradually. Sleep becomes more and more interrupted until it is finally lost altogether. The patient becomes destructive and dirty in his habits. Hallucinations of sight and hearing may be present. He is incoherent, is fed with difficulty, and there is steady failure of physical condition.

2. *Agitated Melancholia.*—Patient is depressed with self-accusations, restless, despondent, and believes that he will never recover. There is often persistent refusal of food. This type is more common in young pregnant women.

3. *Acute Delirium.*—These cases differ from those of acute mania by the severity of both the mental and somatic symptoms. The mental condition resembles that of acute delirious mania. There is marked fever,

hallucinations of sight, refusal of food, constant excitement, insomnia, great prostration and exhaustion, and the termination is often fatal.

4. *Delusional Insanity.*—Delusions of persecution with the belief that they are being annoyed may develop in cases of chronic chorea.

5. *Stupor.*—The mental hebetude and inattention that is so common in choreic patients may become more profound, and they pass into a stuporose state. The memory is commonly very defective in these cases.

Morbid Anatomy.—There are at present no definite macroscopical or microscopical changes known, that can be said to be the actual cause of choreic insanity.

Prognosis.—The prognosis is usually good in the mania and agitated melancholia of chorea. With acute delirium the condition is much more serious and the prognosis must be always guarded, but when the patient once begins to improve there is seldom a relapse.

Delusional insanity or stuporose cases are commonly chronic.

Treatment.—This is largely upon general lines. Good feeding with large quantities of milk and eggs, custards, and solid food, if possible. The more acute the attack the greater must be the amount of food given. If nourishment is refused, artificial feeding must be resorted to. Stimulants are usually required in severe cases. The bowels must be kept open, and salines are recommended for this purpose. For violence the patient must be placed upon a mattress on the floor and surrounded by other mattresses. For persistent insomnia hypnotics must be used. Sulphonal, 30 grains for a child over 13 years; for adults this can be repeated morning and evening if necessary. In agitated cases morphia bimeconate is often useful. Chloral is also recommended.

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The choroid is the posterior part of the *tunica vasculosa*, the anterior part being formed by the iris and ciliary body. In its extent it reaches from the ciliary body to the optic disc, and lies between the sclerotic externally and the retina internally. On microscopic section it presents, from without inwards, the following layers:—

1. *The lamina fusca.*—This consists of lamellæ of loose connective tissue, containing branching pigment cells; it adheres to the sclerotic when that is separated from the choroid, and therefore it is sometimes described as belonging to that tissue.

2. *The lamina suprachoroidea* is similar in structure to the lamina fusca, being composed of lamellæ of branched pigmented connective-tissue corpuscles and a network of elastic tissue. The space between this layer and the last is lined by endothelium, and is considered to be a lymph space.

3. *The lamina vasculosa* consists of a dense network of large intercommunicating veins so closely connected that in some parts the intervascular spaces are less in diameter than the veins themselves. This venous plexus ends abruptly at the ora serrata. These veins are the tributaries of the venæ vorticosæ. Within the fenestrations, which are chiefly fusiform in shape, are pigmented connective-tissue

corpuscles, the presence of which renders this membrane uniformly dense and reflective in most eyes; a deficiency or excess of these pigment-cells will cause the outline of the choroidal vessels to be seen.

4. *The chorio-capillaris*, or *membrane of Ruysch*, is a reticulated vascular membrane of closely intercommunicating capillaries of large diameter; the meshes of this network are small. Here the venæ vorticosæ begin in capillary whorls, the *stars of Winslow*. There are no pigment-cells in this layer, and few, if any, round cells. A delicate structureless membrane, the *elastic layer of Sattler*, is supposed to exist between this and the former layer. This Sattler believes to be the remains of the *tapetum*, a definite layer found in the choroid of certain animals. In carnivora the tapetum is composed of endothelial cells containing minute crystals, producing a shining appearance in the dark. In other animals it is fibrous in nature, and, since the retinal pigment is absent from certain spots, interference of light is produced, causing a similar appearance.

5. *The lamina vitrea*, or *membrane of Bruch*, is an hyaline membrane. It is continued forwards into the ciliary body and iris. It supports the pigmentary epithelium of the retina, and just as this layer, formerly thought to belong to the choroid, has been shown to be epiblastic in origin, so the lamina vitrea is in all probability a layer of the retina, and not of the choroid, being produced originally from the cells of the pigment layer of the retina.

The choroid is almost completely supplied by the *short posterior ciliary arteries.* They are ten or twelve in number, and pierce the sclerotic close to the optic nerve, passing through the lamina fusca into the deeper part of the lamina suprachoroidea; they divide dichotomously, and pass into the capillaries of the chorio-capillaris. Except in the region of the optic nerve, where a circular arterial anastomosis exists around the disc with small branches of the arteria centralis retinæ, the anastomosing branches being called the *cilio-retinal arteries*, the branches anastomose little with each other. Anteriorly they receive a few communications from the arteries of the ciliary region.

The *veins* of the choroid anastomose very freely together; they do not accompany the short posterior ciliary arteries, but are arranged in curves, *venæ vorticosæ*, as they converge to four or five principal trunks; these pierce the sclerotic very obliquely about half-way between the optic nerve and cornea to join the ophthalmic vein.

The *lymphatics* of the choroid help to regulate the intra-ocular pressure. Between the lamina fusca and the lamina suprachoroidea is a lymph space, which communicates by means of perivascular sheaths surrounding the venæ vorticosæ with the lymph space within the capsule of Tenon.

The *nerves* of the choroid are derived from the long ciliary branches of the nasal branch of the first division of the fifth nerve, and from the short ciliary branches of the lenticular ganglion. They are chiefly vaso-motorial in action, and form in the lamina suprachoroidea a plexus, in the meshes of which ganglion cells are found.

THE APPEARANCE OF THE HEALTHY CHOROID must be carefully studied before we can diagnose pathological conditions.

The colour of the *fundus oculi* seen by reflecting light from the ophthalmoscope is due to the following causes:—(1) The blood contained in the chorio-capillaris and lamina vasculosa; (2) the pigment-granules contained in the pigmented layer of the retina, the interstices of the vascular layer of the choroid, and the lamina fusca and lamina suprachoroidea; and (3) the sclerotic, which reflects a certain amount of light through the retina and choroid.

When pigment is altogether absent from both retina and choroid, as in *albinos*, we get a light yellowish red colour reflected from the blood within the capillaries; whilst the interstices between the latter are seen to be of a lighter, almost white appearance, owing to the reflection from the sclerotic beyond the lamina fusca, and thus a fairly well-defined outline of the choroidal vessels is obtained.

In *fair persons*, where the pigment-granules contained within the cells are of a faintly brown colour, the fundus is yellowish red, and the vessels of the choroid can often be seen, although less distinctly than in albinos.

In *moderately dark persons* this pigment becomes of a deeper brown, and all details of the choroid are hidden, the fundus presenting a light brownish-red colour, with no visible choroidal vessels. If, however, as is occasionally the case, pigment is scarce in the pigmented layer of the retina, while abundant in the choroid, a network of red vessels is seen upon a dark background, the so-called *choroide tigreé.*

In *negroes*, and all *dark races*, the pigment is so abundant as to prevent almost all red choroidal reflex, the fundus assuming a brownish gray, or even a slate colour.

The colour of the fundus varies very much with the intensity and colour of the light used, with the state of dilatation of the pupil, and with the refractive condition of the eye. It is brighter, *cæteris paribus*, in proportion to the number of rays of light that can be thrown into the eye.

CONGENITAL AFFECTIONS OF THE CHOROID.—Coloboma of the choroid is a congenital deformity, consisting in the absence of a more or less considerable portion of this part of the tunica vasculosa, and is usually found in the lower and internal part of the globe. When examined by the ophthalmoscope it appears of a bright bluish white colour, with clear cut pigmented edges. The surface of the sclerotic often appears very irregular, is crossed by retinal vessels, and frequently has upon it small aggregations of pigment. The extent of the coloboma is very variable; it usually reaches from the edge of the optic disc nearly as far as the ciliary body. It may embrace the optic disc, in which case the latter is changed in appearance, and looks as if it were hyperæmic by contrast with the white area round it. It may occur in the yellow spot region, *macular coloboma;* it may be very localised, normal choroid being seen all round it; it is often accompanied by coloboma iridis. The retina is often involved in the coloboma, though it may be present over the whole site. Occasionally coloboma of the choroid occurs in both eyes, but when unilateral the left eye is most commonly affected. There is a large scotoma in the visual field corresponding to the coloboma, but otherwise the sight in most cases is fairly good. The scotoma is not necessarily absolute, as the retina may be present.

The cause of the defect is probably due to the formation of adhesions between the developing retina and the mesoblast, the latter of which is not differentiated into choroid and sclerotic. This usually takes place in the position of the retinal fissure, and thus accounts for the usual position of the coloboma. It may, however, take place in any part of the retina; hence the occasional occurrence of the coloboma elsewhere.

Albinism, or congenital absence of pigment throughout the body, may be relative or absolute. In *absolute* albinism the pupils and irides appear pink from the reflected light from the choroid, the irides also on account of the blood contained in their vessels. This condition is usually attended with defective visual acuity, photophobia, and nystagmus. Upon ophthalmoscopic examination, the choroidal vessels are seen most distinctly as a pink fenestrated membrane upon a pale, almost white, background. The hair is usually white throughout the body. In *relative* albinism, the hair is a pale straw colour; the irides present a pale purplish colour, and do not completely shut off the choroidal reflex; the symptoms also are less marked. There is a tendency to acquire pigment as the child grows; the improvement, however, is rarely more than a change from the absolute to the relative condition. By way of treatment, any existing error of refraction must be corrected, dark glasses should be worn, with or without a diaphragm, to cut off some of the light. Peripheral tattooing of the cornea has been performed for the same object.

Congenital crescent is a peculiar grayish white crescentic patch immediately below or, in rarer instances, to the outer side of the optic disc. The disc, excluding the crescent, is usually oval, but with it appears circular or slightly oval in the opposite direction. These crescents present no marginal pigment, which is so often seen in myopic crescents; besides, the latter are usually to the outer side of the disc. It is very probable that this crescent is a partial coloboma of the choroid.

CHOROIDITIS.—Inflammation of the choroid is accompanied by infiltration and exudation, which may be reabsorbed with or without atrophic changes, or may pass into the stage of suppuration. If the morbid process be limited to the choroid proper, it is frequently unattended by external signs of inflammation, being recognised chiefly by visual troubles and ophthalmoscopic appearances. When the other portions of the uveal tract —the ciliary body and iris—are involved in the process, as is usually the case in acute and suppurative forms, the external signs of these inflammations are always present. The two chief varieties of choroiditis are plastic or exudative choroiditis and suppurative choroiditis. Besides these, we have forms of choroiditis accompanied by inflammation of neighbouring parts; these are—irido-choroiditis, retino-choroiditis, and sclerotico-choroiditis.

PLASTIC OR EXUDATIVE CHOROIDITIS.—Etiology.—The most common cause is syphilis, inherited or acquired. In the acquired form it usually occurs towards the end of the first year after the primary infection, or during the beginning of the second year. Inherited syphilitic choroiditis is commonest between the ages of six months and three years; it may occur, however, much later together with interstitial keratitis. Other causes of plastic choroiditis are tuberculosis, gonorrhœa, the simple and profound anæmias, and menstrual disorders. Many chronic cases occur to which no definite cause can be assigned.

Symptoms.—Plastic choroiditis is the most common form, and presents itself under several forms, the symptoms varying with the extent of the area affected, and with its position with regard to the macular region. When unattended with cyclitis or iritis there are no external signs of the disease. *Subjective symptoms* are usually the earlier and more important indications of the disease, but they may be completely absent although the ophthalmoscope shows gross changes. The patient complains of seeing lights of a pale blue or red colour—*phosphenes*—at night, when the eyes are closed and the room is dark. In the daylight he sees large floating specks, especially when looking at a white object; these are larger than the ordinary muscæ volitantes, and tend to obscure the vision by settling upon one portion of the object looked at. Distortion of the outline of objects—*metamorphopsia*—is also a characteristic feature, especially when the exudative change is situated in the macular region; if parallel straight lines are held before the eye they will appear curved in various ways. *Micropsia*—objects appearing smaller than normal—and *macropsia*—objects appearing too large—are symptoms sometimes present. The patient may complain of dark spots in the visual field—*positive scotomata,*—or these may only be found by carefully testing the field with the perimeter—*negative scotomata.* It will then be found that its whole area is not intact, but that, according to the position and extent of the disease, there will be small and large areas in which vision is either defective or altogether absent—*relative* or *absolute scotomata.* The light sense is also appreciably diminished, especially when there is cloudiness of the vitreous. In some cases the patient will complain of a dull aching pain at the back of the eyes.

Ophthalmoscopic Signs.—Recent patches of choroiditis appear as yellowish, ill-defined areas, lying beneath the retinal vessels upon and in the red background of the choroid. These patches may be more or less obscured by a hæmorrhage from a choroidal vessel, this being sometimes the earliest sign of the disease. The yellow exudation may entirely disappear after some weeks, leaving, in rare cases and under proper treatment, the choroid intact; but more commonly, as it disappears, the affected area is found to be more or less atrophic, gradually becoming whiter, with the choroidal vessels showing up, pigment at the same time appearing round each patch and within some of them; crystals of cholesterin may be seen with some of the patches. Still later the choroidal atrophy presents the appearance of white patches, ringed by pigment, with no traces of choroidal vessels, but crossed uninterruptedly by the retinal vessels. It is not uncommon to find both old and recent patches in the same eye. The inflammation usually attacks the adjacent retina; indeed, the exudation often extends through the retina into the vitreous. The vitreous is, therefore, often cloudy owing to the presence of fine opacities. This is more particularly the case in syphilitic choroido-retinitis. The nebulous condition of the vitreous not only interferes with the patient's vision, but prevents the details of the fundus from being clearly seen; the existence of cloudiness of the vitreous is, therefore, always suggestive of choroiditis, although hyalitis without choroiditis is sometimes found. Care must be taken to exclude a nebulous cornea, as this will cause a haziness of the fundus very similar to that produced by fine vitreous opacities. On the other hand, a hazy vitreous will make the outline of the optic disc appear blurred, and thus simulate papillitis.

Pathology.—When a recent patch is examined microscopically, we find a cluster of round and fusiform cells in the lamina vitrea and the choriocapillaris, with great engorgement of the vessels, and perhaps one or two hæmorrhages. The pigment-layer of the retina is then unaffected. As the disease progresses the cells of the pigment-layer proliferate, and the part which is immediately opposite to the patch becomes absorbed, giving it a white appearance; the pigment becomes accumulated at the edges of the patch, and the inflammation extends to the layer of rods and cones, and the outer granular layer of the retina, with small-celled infiltration. Later, this inflammatory exudation becomes absorbed, and gives place to cicatricial tissue; but the structures involved—viz. the outer granular layer, the rods and cones, the uveal tract, and the vessels of the choroid—are destroyed, and their place occupied by this new connective tissue.

Varieties.—Many different kinds of plastic choroiditis are found. The following may be mentioned as those more frequently met with: (1) Disseminated; (2) Central; (3) Peripheral; (4) Diffuse exudative; (5) Myopic.

(1) *Disseminated choroiditis* consists of numerous round or irregular spots scattered over the fundus. These pass through the changes mentioned above, and mostly end in patches of complete atrophy. They may become confluent, involving the whole or the greater part of the fundus. The optic nerve may become involved in the process, in which case it becomes first hyperæmic and finally atrophic. This form of choroiditis may easily be mistaken for *guttate choroiditis of Tay* (p. 286).

(2) *Central choroiditis* consists of exudation in the macular region, forming an irregular, more or less circular patch, with considerable

pigmentation. Vision is impaired, and a large central scotoma is present. This is especially frequent in old people, where a very chronic inflammation, without much pigmentation or exudation, is followed by well-defined atrophic changes. In such cases the fenestrated arrangement of the choroidal vessels is clearly seen. This senile form is usually bilateral, and must be carefully sought for before the extraction of all senile cataracts, as its presence will greatly modify the prognosis. Before a diagnosis of amblyopia is made, either congenital or toxic, the macular region should be carefully examined for central choroiditis.

(3) *Peripheral* or *anterior choroiditis* is that form in which only the peripheral parts of the choroid are affected. Owing to the outlying position of the lesion, direct vision is but little disturbed, and the disease may be easily overlooked unless the peripheral portions of the fundus are carefully examined.

(4) *Diffuse exudative choroiditis*, extending over the whole fundus, is sometimes associated with syphilis. Although the whole choroid is affected, yet the deposit of lymph appears to assume the form of circumscribed patches, varying somewhat in tint from a yellowish red to white, with pigmentation according to the stage of the affection. It is always more or less obscured from view by fine dust-like opacity of the vitreous, and not infrequently large membranous floating opacities of the vitreous are present. The vitreous and the retina may ultimately become clear, and then the ophthalmoscope reveals more definitely the large atrophic patches in which masses of pigment are here and there distributed. Such cases, when they have arrived at an advanced stage, are often difficult to distinguish from *retinitis pigmentosa* (see "Retinitis"), for the retinitis secondary to syphilitic choroiditis tends to simulate that disease. Peripheral retinal pigmentation, night-blindness, and contraction of the visual field occur; and it is only by the presence of vitreous opacities, patches of choroidal atrophy, relative distension of the veins, together with perhaps concurrent iritis or cyclitis, and a definite history of syphilis, that the right diagnosis can be arrived at. The visual field, too, may help, for in many instances there is no peripheral contraction, but a ring scotoma, which is almost pathognomonic of syphilitic choroido-retinitis.

This kind of choroiditis almost invariably affects the retina to a considerable extent.

(5) *Myopic choroiditis*, see *Sclerotico-choroiditis.*

Prognosis of Plastic Choroiditis.—Plastic choroiditis runs a chronic course; its worst feature is a tendency to recurrence. Many cases are so chronic that they extend over years without perceptible change; whilst others present new spots of exudation from time to time, until the choroid is ultimately covered with atrophic patches. In bad cases the optic nerve and retina are involved, and partial or total blindness results. Vision, however, may be almost completely restored so long as the macular region is free. Central changes, like peripheral, are attended with subsequent atrophy, which gives rise to permanent and absolute scotomata; consequently, direct vision becomes completely lost. Diffuse exudative choroiditis is invariably attended with gradual failure of sight until complete blindness ensues.

Treatment of Plastic Choroiditis.—It is necessary to find out, if possible, the cause of the disease. A history of syphilis can often be elicited, and should the disease be active, mercury in some form must be administered, either with or without iodide of potassium; the subconjunctival injection of mercury has been warmly advocated by various continental surgeons, and the discussion of this and other methods of treatment will be found under the head of "Iritis." Other constitutional causes require general treatment. Locally, if there is active inflammation, as shown by dull pain, diffuse vitreous haze, and fresh exudations, leeches applied to the temple, diaphoresis by hot-air baths or pilocarpine hypodermically injected, and profuse purges, are necessary. Counter-irritation by means of the Argyll-Robertson method frequently helps to clear up the condition. This consists in rubbing into the skin of both eyelids pure silver nitrate until vesication is produced. The eyes should be protected from the light by tinted glasses; they should be rested as much as possible, and no near work attempted. It may be necessary to atropinise the eyes to prevent accommodation.

Should there be no history of syphilis, it is in many cases still advisable to try the mercurials and iodides, as great improvement is often obtained by their use independent of any syphilitic history. If the general health is run down it must be supported by a nutritious diet, regular outdoor exercise, and by the exhibition of tonics, such as iron, strychnine, quinine, etc.; local treatment must also be adopted as indicated above. It is advisable in most cases of choroiditis to avoid alcoholic stimulants.

SUPPURATIVE CHOROIDITIS is a diffuse suppurative inflammation of the choroid, and is always attended with suppurative irido-cyclitis. There is, in fact, a general inflammation of the tunica vasculosa, which usually spreads to the other tunics of the globe, and gives rise to that condition known as *panophthalmitis.*

Etiology.—The causes of suppurative choroiditis may be divided into three groups:—

(*a*) Injury, of a penetrating nature, if septic infection by pyogenic matter is produced. Operative measures on the eyeball, where due aseptic precautions have not been taken, may be placed under this category.

(*b*) There may be an extension of an inflammation elsewhere, such as from a septic ulcer of the cornea.

(*c*) Inflammation of the choroid may be the result of metastasis. Of metastatic choroiditis the most important is the puerperal form, occurring as a symptom of pyæmia, usually in the second week after delivery. It is also occasionally found in ulcerative endocarditis, cerebro-spinal meningitis, and during the acute specific fevers. It is due to septic embolism of the retinal and choroidal vessels.

The *symptoms* from the first are those of intense inflammation. The eyelids are red, swollen, and œdematous, so that it is with difficulty that the eye can be examined; were it not for the absence of discharge, the condition might be mistaken for purulent conjunctivitis. There is great chemosis and conjunctival injection. The cornea is hazy and anæsthetic; the iris muddy and immovable, with a somewhat dilated pupil. The anterior chamber is shallow and often contains pus (*hypopyon*). There is slight proptosis, and the tension is somewhat raised. If the media are sufficiently clear the exudation into the vitreous can be seen as a yellowish reflex.

As time goes on there may be a gradual subsidence of the symptoms, the proptosis diminishes, the inflammation subsides, the tension falls, and gradually a general shrinking of the whole globe (*phthisis bulbi*) occurs. In the acute form, however, suppuration occurs, with marked constitutional symptoms. High fever, vomiting, and violent pains ensue; the proptosis increases, the lids become more swollen, until the eyeball gives way anteriorly, with escape of pus and disappearance of the acute symptoms.

Pathology.—Suppurative irido-choroiditis is attended with suppurative retinitis; the vitreous also, participating in the progressive and destructive inflammation (suppurative hyalitis), becomes destroyed and replaced by pus, and the eyeball is converted into an abscess-cavity. In such a state either the cornea will slough and the pus be discharged, or the pus will become inspissated by absorption of its liquor puris; in either case the globe will shrink. In many cases the exudation in the vitreous is more puriform than purulent, and the yellowish white reflex seen has a somewhat similar appearance to glioma retinæ. Hence it has been termed *pseudo-glioma.* The puriform exudation often undergoes partial organisation, and so the ophthalmoscopic condition may remain for some weeks after the subsidence of active inflammatory changes; eventually, however, the eyeball is sure to shrink, become puckered and hardened, and in some cases the choroid will undergo ossification.

Microscopically the choroid will be found to be greatly thickened and exceedingly cellular, the cells aggregating into clusters which form small abscesses; these soon coalesce and convert the choroid into a diffuse suppurative tract. The inflammatory changes in the iris, ciliary body, retina, and vitreous are similar to the microscopical characters of inflammation found in other parts of the body. It is advisable to mention, however, that the partially organised exudation in the ciliary body looks under the microscope not unlike a melanotic sarcoma, on account of the complete derangement and multiplication of the pigment-cells. The clinical history, together with the condition of the vessel-walls, will explain, however, its nature.

In metastatic choroiditis it is often possible to find the infecting emboli, which can be shown to contain pyogenic organisms, the streptococci being the most common.

Diagnosis.—The main difficulty is to distinguish between a chronic and subacute form of suppurative choroiditis and certain intraocular growths, of which glioma is much the most common. The diagnosis depends chiefly on the history, the presence, or absence of recent inflammatory signs, and the tension of the globe. Suppurative choroiditis is usually preceded by a perforating injury, or by a severe illness such as a specific fever. Again, signs of recent iritis will be seen; and, lastly, intraocular neoplasms, though at first they may be present in a globe of normal tension, soon produce a rise of tension with all the signs of secondary glaucoma, suppurative choroiditis, on the other hand, causing a fall in the tension. Other points to help will be the age of the patient, glioma only occurring in young children, and the appearance of the reflex, which is of a much more glistening appearance in glioma than in pseudo-glioma.

Prognosis.—The diseased eye is sure to be lost, so far as sight is concerned, and will inevitably pass into a state of atrophy. In all cases the question of sympathetic inflammation in the other eye has to be carefully considered (see "Sympathetic Ophthalmitis").

Treatment.—Local leeching, hot fomentations, combined with morphia, either hypodermically or by the mouth, are useful in allaying pain. As soon as pus is evidently accumulating in or behind the aqueous chamber prompt surgical interference is indicated. If the eye be left to itself there is considerable risk of the inflammation extending along the optic nerve to the brain and its membranes, and so causing a fatal termination. Excision of the globe is, in my opinion, the best and safest way of treating this severe condition, great pains being taken after the enucleation to treat the socket antiseptically. Some surgeons, however, are doubtful as to the propriety of removing an eye whilst in this inflamed and suppurating condition, and prefer first to make an incision through the anterior part of the globe so as to relieve pain, tension, etc., and to give a free outlet to the pus, postponing the excision until the inflammatory symptoms have subsided.

Irido-Choroiditis (uveitis) is an inflammatory condition involving the whole uveal tract. It may be *acute* or *chronic.*

Acute Irido-Choroiditis is either suppurative (see "Suppurative Choroiditis") or sero-plastic (see "Sympathetic Ophthalmitis").

Chronic irido-choroiditis attacks each part of the uveal tract either simultaneously or successively. It is very often preceded by an acute attack of iritis or choroiditis. Subacute exacerbations occur from time to time. Its tendency to relapse is its predominating feature; years may pass away before there is a permanent cessation of inflammatory symptoms. The prevailing clinical features are evidences of iritis, cyclitis, and choroiditis, such as posterior synechiæ or pigment on the lens capsule; thinning of the iritic tissue so that in some cases the dull choroidal reflex may be seen through the reticulum of the iris, as if through a curtain; thinning of the sclera over the ciliary body, from chronic cyclitis, which allows the blue ciliary body to be seen through; eventually a ciliary staphyloma may appear. Numerous and large vitreous opacities are seen; and, if the vitreous is not too cloudy, patches of choroiditis and choroidal atrophy, with general thinning of the choroid, may be made out. Posterior polar cataract or irregular opacities in the lens are not uncommon, and at last the whole lens may become cataractous. Subjective signs are present, namely, defective vision, sometimes amounting to bare perception of light, phosphenes, dull aching pains in the eyes, etc. The tension is usually slightly raised at first, but soon becomes subnormal (T-1 or T-2). The prognosis is unfavourable. The disease is usually symmetrical, and complete blindness will almost certainly ensue sooner or later.

Treatment.—Little can be done beyond treating the symptoms and supporting the general health. Iodide of potassium assists in some cases, chiefly on account of its alterative action and its powers in aiding the absorption of all chronic inflammatory exudations. Mercurial inunctions are also useful for the same purpose. Iritis must be treated in the early stage by instilling atropine. Increase of tension is rarely present, and certainly is never sufficiently pronounced to necessitate operative interference.

Retino-Choroiditis.—There is almost always some accompanying retinitis with all cases of plastic choroiditis. It is unnecessary to add anything to what was said under that heading.

Sclerotico-Choroiditis, or Myopic Choroiditis.—This is rather of the nature of an atrophic condition of choroid than a true inflammation, and will be referred to under Myopia.

Tuberculous Disease may affect the choroid in three ways, which differ in ophthalmoscopic signs, prognosis, and treatment.

(*a*) *Tuberculous Disseminated Choroiditis.*—This form of chronic inflammation of the choroid is very similar to the syphilitic variety, and is, according to some authorities, as common. It is with difficulty distinguished, but the patches are smaller and slightly raised. Just as in the syphilitic form, degeneration takes place with choroidal atrophy, the process being, however, slower. Its treatment is unsatisfactory; before atrophic changes have set in mercurial inunction may help absorption of the exudation, and phosphates or arseniates with cod liver oil should be prescribed by the mouth. The prognosis is not so good as in the syphilitic variety.

(*b*) *Miliary Tuberculosis of the Choroid* occupies the region of the chorio-capillaris and the vascular layer, and is quite behind the uvea. It is most commonly found in cases of acute miliary tuberculosis, but it may be present in all forms and stages of tuberculous disease.

Ophthalmoscopically it appears as a grayish hemispherical eminence varying from 3 mm. to 1 mm. in diameter, and may be even smaller. One or several of these first appear in the macular region, and are afterwards followed by others in the neighbourhood. The youngest tubercles are very small; the oldest are the largest, and are somewhat white at the centre. The ophthalmoscopic signs are very similar to those of disseminated choroiditis, though the two conditions are not likely to be mistaken owing to the different general conditions. The patches are more raised, rarely pigmented, more clearly defined, and less brilliantly white. In the form that occurs in acute general tuberculosis they appear as a rule only a short time before death. Papillitis is a frequent accompanying symptom. Both eyes are nearly always affected.

Microscopically each patch shows the typical structure of tubercle, though the specific bacilli are not always to be found.

In cases of acute tuberculous disease where there are typhoid symptoms, and in tuberculous meningitis where the diagnosis is not always easy, the detection of tubercles of the choroid is of great assistance in clearing up the case, although the absence of choroidal tubercle does not prove the absence of tuberculous disease in other organs.

(*c*) *Tuberculous Tumour of the Choroid.*—This condition is probably always secondary to tuberculous disease elsewhere, though this cannot always be proved. In early stages, a retinal detachment, less defined than the detachment due to choroidal sarcoma, will be the only sign. As growth takes place glaucomatous symptoms rarely occur, the coats of the globe rapidly giving way with the formation of a staphyloma and subsequent panophthalmitis. It is doubtful if general infection can occur, so enucleation should be postponed till vision is lost. The growth is always unilateral.

GUMMA OF THE CHOROID is very rare. It may exist in conjunction with gumma of the iris or ciliary body. The diagnosis is necessarily difficult, inasmuch as the vitreous is hazy and the fundus cannot be seen. It rests mainly on the concurrent syphilitic lesions, the history, and the effect of antisyphilitic treatment. Sight is usually greatly impaired, but, if the condition is attacked in time, almost perfect vision may be regained.

SARCOMA OF THE CHOROID is the commonest intra-ocular tumour of adult life, just as glioma is the most frequent in infancy and early childhood.

Etiology.—No cause has been discovered. There appears to be no relationship between it and blows on the eyeball.

Symptoms.—Sarcoma of the choroid usually begins in a manner so insidious as to be unnoticed even by the patient until the tumour has attained a considerable size; even then it is often discovered accidentally. Sometimes, however, though rarely, the growth of the sarcoma is accompanied by local pains, phosphenes, etc.

When seen at an *early stage* there may be nothing externally to attract notice. In addition to the dimness of sight which may have first caused the patient to apply for advice, we find that the visual field is defective, and when it is examined by means of the perimeter, presents a scotoma corresponding to the position of the tumour. With the ophthalmoscope the outline of the tumour can sometimes be seen to form a rounded prominence, pushing the retina forwards into the vitreous cavity. In this early stage inflammatory signs are absent, and the tension is normal or even slightly subnormal.

At a *later stage* the presence of the tumour is accompanied by a distinct increase in the tension of the globe, and the eye presents other symptoms of glaucoma. The anterior ciliary vessels are congested; the cornea becomes hazy and more or less anæsthetic. The anterior chamber gradually becomes shallow. The iris is sometimes subacutely inflamed, and forms posterior adhesions (*synechiæ*) to the capsule of the lens, which render the pupil irregular. Not infrequently the iris is atrophied, and it may be detached at that part of its periphery which corresponds to the position of the tumour. The vitreous also is frequently rendered cloudy by the presence of opacities. The vision has gradually become worse, and is now reduced to bare perception of light. When the disease has progressed so as to destroy vision there is frequently considerable pain in the ciliary region and lachrymation, more severe than in true glaucoma.

In the *third stage* the tension of the globe is suddenly reduced, signifying that the sclerotic has given way, and the glaucomatous symptoms are relieved. The growth, however, now rapidly extends and infiltrates all the surrounding structures.

Pathology.—Sarcoma of the choroid may be divided into the following varieties:—

A. Leuco-sarcoma — (*a*) Spindle-celled; (*b*) round-celled; (*c*) mixed-celled.

B. Melano-sarcoma — (*a*) Spindle-celled; (*b*) round-celled; (*c*) mixed-celled.

C. Intervening grades of pigmentation.

Of these, spindle-celled melano-sarcoma is the commonest. Leuco-sarcoma is found in about one case in every eight or nine, but even then a few pigment-containing cells are present. When the growth is white the cells are usually of the round variety, and it appears probable that this form starts from the non-pigmented chorio-capillary layer, while the melanotic form has its origin in the deeper pigmented layers.

The tumours usually are firm; they generally contain some blood-vessels, and sometimes are very vascular; the walls of the blood-vessels are composed of sarcomatous elements. They have an even and smooth convex surface so long as the lamina vitrea remains intact. When they perforate this membrane they grow more rapidly, become hour-glass in shape, and present an irregular granular surface. Sometimes there is also effusion of serum or blood beneath the retina. They may increase so as to fill the whole globe and distend its walls before invading the extra-ocular tissues; but in many cases the tissues outside the sclerotic are affected by the new growth, whilst the tumour within the globe is quite small; in these cases the cells pass to the outside by means of the sheaths of the blood-vessels, which are seen to be thickened and altered by the presence of cells similar in character to those of the tumour.

The neighbouring lymphatic glands are not affected, but secondary sarcoma is liable to be set up elsewhere by a process of embolism, the cells being conducted from the primary source by the blood-current. The liver is the organ usually first affected.

Diagnosis.—It is obvious how important an early diagnosis is, but this is often extremely difficult. In the early stage there is always great doubt, whether the ophthalmoscopic appearance of the retina is due to a simple detachment from subretinal effusion or to a sarcomatous growth in the choroid. When the latter is the case the detached retina may retain some colour or be pigmented; it may occur at any part of the fundus, and it does not flap about when the eye is moved. In simple detachment the detached portion is bluish white, usually occurs at the lower segment of the fundus, and may flap about freely when the eye is moved. Occasionally a vascular network of the sarcomatous growth can be detected through the retina. In any case of extensive detachment occurring in one eye only, and when there has been no myopia or history of injury, we must suspect sarcoma of the choroid.

In the stage of increased tension sarcoma of the choroid may easily be mistaken for acute primary glaucoma. If the fundus can be seen there is usually no difficulty; but if this is impossible, the diagnosis must be made from the history of the case, whether any symptoms of glaucoma had previously existed, and by the condition of the visual field and the projection of light. An important point in the history is the relation between the onset of pain and the loss of acuity of vision. In primary glaucoma the two are almost synchronous, whereas in choroidal sarcoma the sight in most cases has been gradually getting worse previous to the glaucomatous symptoms.

Course and Prognosis.—If left alone the disease usually takes some years to run through all its stages, the end being always fatal, the patient dying generally from extension into the brain or from metastasis. In all cases, however early the diagnosis is made, the eye is lost, and we can never promise that there will be no recurrence, local or general.

Treatment.—Enucleation should be performed as early as possible, together with excision of 4 or 5 mm. of the optic nerve. If the growth has extended outside the globe the orbit should be emptied.

CARCINOMA OF THE CHOROID is very rare, and is always secondary, usually to carcinoma of the breast. Death usually occurs within twelve months.

CHOROIDAL DEGENERATION.—*Guttate choroiditis of Tay* is characterised by a number of yellowish-white specks usually, though not necessarily, in the macular region. They are probably spots of colloidal degeneration of the choroid.

Ossification of the choroid frequently follows phthisis bulbi. It may be extensive, a thin plate being present on the inner or vitreous part of the choroid; or there may be merely a few spicules of bone scattered throughout the choroid.

INJURIES TO THE CHOROID.—Rupture of the choroid is always the result of external violence, such as a blow or a fall. The accident is usually followed by hæmorrhage into the vicinity of the wound, causing opacity of the vitreous. After a few days, as the blood becomes absorbed, we can see a whitish line in the fundus, with a little blood clinging to its edges. It is usually curved, concentric with the edge of the disc. Subsequent to the rupture there is frequently a tendency for masses of pigment to appear round the exposed sclera.

Concussion Choroiditis.—Rupture of the choroid is often accompanied by macular choroidal degeneration.

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Chromidrosis. *See* Sweat Glands.

Chyluria. *See* Filariasis, Urine.

Cicatrices.

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Cicatrices or scars are new formations of connective tissue which develop in place of losses of substance.

Ordinarily, however, the terms are applied to superficial formations of "scar tissue" which are covered with newly developed epidermis.

A healthy cicatrix has a smooth, more or less glossy surface. In its earlier stages it is of a pinkish colour, due to the persisting vascularity of its connective tissue; later it becomes white, dense, and much less vascular. It differs from true skin in being less elastic and in possessing no surface furrows, hair follicles, sebaceous or sweat glands. The surface shows slight longitudinal ridges, caused by contraction.

The size of a scar depends on the amount of normal tissue which has to be replaced.

A. When healing takes place by *first intention*, the resulting cicatrix is "*linear*," and in time it tends to become less and less apparent.

Under the microscope a transverse section shows a narrow non-vascular band of connective tissue covered by epithelium, and with its elements arranged parallel to the surface.

B. When healing takes place by *second intention*, the resulting cicatrix has a more widespread surface. Clinically, its characters depend on the nature of the preceding granulation tissue.

Microscopically the cicatrix of a wound which has healed by second intention shows on the surface a thin covering of epidermis. This covering is indented from beneath by papillary tufts, which are simply the organised vascular tufts of the preceding granulation tissue. They contain no tactile end-organs, the connective tissue forming them is denser and less vascular than in the case of the papillæ of normal skin, and they tend in time to become more flattened. Below this is fibrous connective tissue, arranged in interlacing bundles, more or less vascular, and more or less cellular, according to the age of the scar. Unna states that elastic tissue may in time be re-formed in the scar. At the margin of the cicatrix its structure rather rapidly merges into that of the surrounding normal tissues.

Cicatrices are divided into three varieties:—

1. Normal or flat cicatrices, whose level is the same as that of the surrounding skin.

2. Atrophic cicatrices, in which over-contraction of the fibrous tissue has taken place, and the surface is retracted below the level of the surrounding skin.

3. Hypertrophic cicatrices, where in consequence of over-reaction, or long continuance of the healing process, an excess of scar tissue has been formed, so that the level of the cicatrix is higher than that of the surrounding skin.

Treatment of such cicatrices is only necessary when they cause disfigurement, particularly on exposed surfaces. Measures directed towards keeping up their vascularity, such as *massage or painting with iodine*, tend to induce a degree of resorption, or if the surrounding skin is lax, the scar may be excised and the edges accurately apposed with the view of substituting a linear cicatrix.

PATHOLOGICAL CONDITIONS OF CICATRICES

1. WEAK CICATRICES are thin, shining, reddish, easily wrinkled and cracked, prone to break down and ulcerate in the centre, the friction of the clothes being sufficient to cause this.

They result when the original wound has been very extensive and contraction has been incomplete during the healing process. Hence they occur after severe burns and scalds, particularly if these have affected the tissues deeply; but a smaller cicatrix when it is adherent to bone may also show a weak tendency.

Certain constitutional conditions, as tuberculosis (scrofula), anæmia, syphilis, scorbutus, conduce to the formation of weak cicatrices. The weakness may be due to defective granulation, or defective epithelial formation (keratinisation).

Treatment.—Treat locally, as for ulcers. In addition to topical applications, the part must be carefully protected from the slightest injury and the general health attended to, treating the constitutional condition if it can be made out.

If the wound has been extensive, there is less chance of good cicatrisation, and a plastic operation is necessary. Thiersch-grafting may not give a good result in such an ulcer, and it is better, after scraping or cutting away all the unhealthy tissue, to transplant a piece of skin from the neighbourhood (*vide* "Skin-grafting").

2. EXUBERANT, HYPERTROPHIC, DEFORMED CICATRICES.—Cicatrices of ragged wounds, such as are caused by glass, splinters of wood, or nails, are generally prominent and ugly. The scars of tuberculous ulcers are extensive and irregular. Wounds which take a long time to heal by granulation may develop a large hypertrophic cicatrix. Treatment is required only when the scar is on an exposed surface, and then it is for the removal of the disfigurement caused.

When the surrounding skin is tense, operative interference is inadmissible, though some improvement may be brought about by measures, such as massage, painting with iodine, etc., directed to the increase of vascularity in the connective tissue; but if the skin be lax, the scar may be completely dissected out, the edges accurately apposed with buried catgut sutures, and a linear cicatrix substituted for the hypertrophic one.

3. CONTRACTING CICATRICES, CAUSING DEFORMITY.—Such cicatrices may in themselves be quite healthy, but they cause conditions which become pathological.

(*a*) *Simple contracting Cicatrix.*—The amount of contraction depends on the depth of the original wound. When the whole cutis has been destroyed, the contraction is greater than when only the papillary layers or part of them are destroyed.

The cicatrix is broad; it is thin and parchment-like, of a light colour, mottled on the surface. The surface is uneven, and marked with ridges. Such a cicatrix may be seen after extensive burns of the front of the neck and thorax, and causes dragging down of the jaw, eversion of the lower lip, and constant dribbling of saliva, which is apt to cause superficial ulceration of the scar. If on the face, they cause ectropion or anchylosis of the jaw, and when in the neighbourhood of the natural orifices have caused their distortion or partial closure.

When occurring in flexures they tend to cause adhesions in the angle, and the parts become firmly connected by a strong prominent cicatrix, the so-called *vicious bridle.* Examples of such cicatrices are seen in the axilla—connecting the arm to the trunk, in the bend of the elbow, causing persistent flexure, or in the hands and feet, causing distortion or adhesion of the digits.

If possible, treatment should be begun early in cicatrisation, before contraction has taken place, by splints and extension apparatus.

If cicatrisation is complete, treatment usually consists in dividing the cicatrix completely, either transversely or in a V-shaped manner, stretching it and filling up the gap thus caused by skin-grafts. In making the incision it is well to remember that important nerves and vessels may be involved in or adherent to the cicatrix. Simple extension, compression, and massage of the cicatrix or painting it with iodine have also been recommended with the view of increasing vascularity and promoting absorption, but such measures are never wholly successful. Hebra recommends subcutaneous injection of a 15 per cent alcoholic solution of thiosinamin, but experience of this remedy is limited.

(*b*) In the *depressed, adherent scar*, treatment consists in dividing the band of attachment, by means of a tenotome passed in from the edge, and raising the scar. Less active measures, with a view to increasing the vascularity and promoting absorption, are never wholly successful.

When it results from a scrofulous (tuberculous) gland, Adam's operation (*Brit. Med. Journal*, 1876) may be employed. After freeing the cicatrix from the side, he passes two hare-lip pins, at right angles to each other, under it so as to raise it. After three days, during which the scar tissue has become swollen and succulent, and the space beneath filled with blood clot, he removes them. The scar subsides to the level of the skin without re-forming adhesions.

Cicatrices may also cause deformity by yielding to pressure from beneath, as in those following laparotomies, when ventral hernia may result.

4. Painful Cicatrices.—Pain may be present in any cicatrix, but is most common in those of amputation wounds. The pain is caused by inclusion of the nerves in, or their adhesion to, the cicatricial tissue. Some authors say that the pain is caused by neuritis dependent on the contraction of the scar.

Neuralgia of the Cicatrix.—In this case the pain, which is paroxysmal, is generally due to some constitutional condition affecting a cicatrix in itself healthy. It may persist in spite of operative interference.

Treatment.—Free adhesions in case of depressed or adherent cicatrices. If the cicatrix be small, excise it altogether and slide a flap of skin over it. If the cicatrix be extensive, neurotomy, neurectomy, or nerve-stretching may be useful.

Where there is no abrasion of the surface, in the case of a small, painful cicatrix, ointment of aconitine (1 gr. to ℥i.) is of use as a palliative; or, if broad, it may be protected by simple plaster, or emplastrum opii, or may be simply covered with cotton wool.

5. Discoloured Cicatrices.— The cicatrices from old chronic and varicose ulcers have always a permanent brown colour. Large scrofulous cicatrices have an unsightly livid tint.

Foreign matter may be present, such as unconsumed grains of gunpowder in cases where the injury has been caused by an explosion, or in tattooing.

Occasional corneal opacities are due to the presence of reduced silver, where the preceding ulcer has been treated with silver nitrate.

These discolorations occasionally require treatment when on exposed surfaces.

KELOID, OR CHELOID

Is a comparatively rare fibrous new growth of the skin which usually develops in an old cicatrix. It was first described by Alibert in 1810. Keloid was formerly divided into true or spontaneous and false or cicatricial keloid, but this distinction is no longer generally held, as most of those termed spontaneous probably develop in minute scars, such as are left by acne pustules. Histologically they are identical.

The etiology is practically unknown. Its growth seems to be dependent on some constitutional state; for example, it is very frequently associated with the tuberculous diathesis. It is more common in negroes than in white men, and it may occur in more than one member of the same family. All grades of society are liable to it, both sexes, all parts of the body, and it may occur at almost all periods of life. Morrow states that in his experience it is more common between 30 and 50 years.

Keloid often develops in scars which have been unduly irritated, and it may develop in a healthy scar long after this is fully formed.

In individuals liable to it, it develops after such slight operations as piercing the ear for ear-rings, extirpation of warts, cauterisation, or after leech and insect bites, or on the sites of acne pustules. It may be multiple, as in a case of Schwimmer's quoted by Morrow, in which 105 tumours were counted.

Keloid is a scar-like growth, usually circumscribed and isolated. It may occur as a rounded, firm, pinkish elevation, with irregular or smooth surface, and on section creaks under the knife. Often from the central mass claw-like processes extend in the surrounding skin. As a rule it has the shape of the scar from which it has developed, but occasionally a part only of the scar undergoes the morbid change.

Neumann, Warren, and Kaposi have described the microscopical appearance.

Connective tissue begins to develop from the walls of the small blood-vessels of the cutis. In this way dense fibrous tissue is formed under the epithelium, the bundles being placed chiefly parallel to the surface, though a few small bundles are perpendicular to it. This is more vascular than true scar tissue. The epithelium is thinned over it, and the germinal layer of the rete mucosum becomes irregular. The growing part of the tumour is more cellular than the central older part, which may become very firm, dense, and hollowed on the surface. Frequently dilated veins course over it.

The tendency to recurrence is explained by the development of spindle cells in the adventitia of the vessels—a condition often extending to some distance from the edge of the tumour.

Keloid is usually confined to the dermis, leaving the papillary layers intact. If of a large size it may cause deformity by contraction. It may project any distance from a line to ¼ to ½ inch above the level of the surrounding skin.

Clinically, keloid starts near the edge of the scar; it may cause no symptoms other than a sense of weight. There are usually, however, subjective symptoms such as itching or sensitiveness on pressure; pain is not common; ulceration and hæmorrhage may take place.

It may develop in a few weeks, or at most a few months, after the exciting cause. Its natural tendency, as of all young fibrous tissue, is to contract after a time and atrophy.

According to Neumann keloid never develops malignancy.

Diagnosis.—From hypertrophied cicatrix by its outline, elevation, consistence, colour, and presence of itching or pain, and by the tendency of the latter to become stationary.

The diagnosis from scleroderma and Addison's keloid (morphœa) will be discussed under "Scleroderma," and from rhinoscleroma, under "Skin, Benign Tumours."

Treatment.—Local treatment is directed towards decreasing the vascularity of the tumour. Applications like collodion (not flexile), either alone or containing lead acetate, a drachm to five drachms of collodion, are useful. Compresses, strapping, elastic pressure, must be applied with caution, as they are apt to cause sloughing, and are only applicable when the keloid is circumscribed, painless, not greatly projecting, and resting on a bony surface. They are especially contra-indicated in diabetics and delicate patients. Multiple scarifications are of doubtful benefit, either alone or followed by mercurial ointments. In a few cases strong salicylic acid plasters 10 to 20 or 25 per cent have been successful.

Itching is best relieved by regulation of the diet, by an occasional saline purgative, Fowler's liquor arsenicalis, liquor potassæ in large doses, or by frequent tepid bathing, either simple or with addition of carbonate of soda or potash, common salt or bran.

Pain is relieved by ointments of camphor and chloral.

Surgical interference by excision is only justifiable when the surgeon is reasonably certain that he can improve the appearance of the part by substituting a linear scar for the keloid, or at least of lessening its size.

Tendency to recurrence is very marked, and the recurrence is often more exuberant than the original growth. In most cases it is best to leave well alone, and employ only palliative and constitutional remedies.

Excision may be contemplated when the keloid is very unsightly and where there is plenty of free skin surrounding, so that the edges of the wound can be readily apposed without tension. The growth must be completely removed, and buried sutures used, approximation being assisted by strips of gauze fixed to the skin with collodion. Everything must be done under strict asepsis.

Constitutional Treatment.—Cod liver oil, iodide of iron, nourishing diet, are of use where tuberculosis is suspected; mercury and the iodides in syphilis. Good results have been obtained in the treatment by sea bathing.

MALIGNANT DISEASES OF CICATRICES.—Sarcomata develop rarely in cicatrices. More commonly, but still rarely, epitheliomata occur. They may develop at any period after the cicatrix has formed. One case is on record where epithelioma developed in an old lady in the scar of a burn which she received when a child of four years.

Epithelioma develops very slowly in the cicatrix, and may not involve neighbouring glands until it grows beyond the cicatrix into normal tissue, in which it develops more rapidly.

Treatment consists in early and complete excision.

LITERATURE.—1. ASHHURST. *International Encyclopædia.*—2. FORGUE-RÉCLUS. *Traité de thérapeutique chirurgicale.*—3. MORROW'S *System.*—4. UNNA. *Histopathology of the Skin.*

Ciliary Body. *See* IRIS.

Circumcision.

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CIRCUMCISION is the operation for the removal of the prepuce in its whole circumference. Its origin is obscure. As a religious ordinance it dates from the time of Abraham, but there are proofs of an earlier Egyptian origin, while it was undoubtedly practised in early times in Central America, New Zealand, and the South Sea Islands. At the present day it is not only a Jewish ritual performed in infancy, but is customary at puberty among Mohammedans, and prevails in some parts of Central Africa.

Apart from the question of religion and custom, one may say, generally, that circumcision is indicated where there is a condition of phymosis, that is, such a narrowing of the prepucial orifice where the cutaneous and mucous membranes meet, that retraction and reposition are difficult or impossible. Difficulty in retracting the prepuce from epithelial adhesions to the glans is normal at birth, and is not phymosis, and only becomes so when natural or artificial means fail to relieve the condition. Operation is, therefore, only indicated when the normal process of retraction is delayed, and the orifice narrowed by slight recurring attacks of balano-posthitis, and more especially if complications arise which experience has proved to depend upon this condition. The phymosis may be partial or complete: hypertrophic when the outer layer of the prepuce is elongated; atrophic when tightly stretched over the glans, inflammatory, œdematous, or senile.

The complications arising from phymosis are many: they may take the form of urinary troubles, local inflammation and irritation, a frequent combination of the two, and maldevelopment of the penis and glans. Of urinary troubles there may be frequency of micturition, straining and pain, eneuresis, retention, incontinence, and overflow, dilatation of the bladder, ureters, and kidneys, with, as a consequence, prolapsus ani, hernia, hæmaturia, uræmia, cystitis, and stone. From local inflammation and irritation arise pain, swelling, œdema, muco-purulent discharge with increase of the phymosis, erections, masturbation, seminal emissions, and other disturbances of the sexual functions, leading to nervous troubles, gastralgia, palpitation, hypochondriasis, and it is even said simulated or real disease of the hip-joint, epilepsy, and brain affections. A combination of the two naturally aggravates both, while later in life maldevelopment of the penis and glans, difficulties in coitus, and the liability to venereal disease and epithelioma have to be considered. Although all conditions of true phymosis are really acquired, as proper treatment in infancy would have prevented them, still for convenience, those occurring in children whose foreskins have never been retracted are termed congenital, while those which appear later in life, where the prepuce has previously been easily retracted, are called acquired. Acquired phymosis can only occur when the prepuce is long, as it normally is in 33 per cent of adults. The causes are usually inflammatory, from gonorrhœa, chancres, eczema, or herpes; but senile phymosis depends on atrophy of the glans and limb of the penis, with chronic irritation.

Should one or several of these conditions exist, the next question to consider is when is operative interference necessary, and regarding this the most varied views prevail.

According to some, every long prepuce, whether it can be retracted or not, demands operation, as it may at a subsequent date lead to trouble. The other view is, that, as in the majority of cases, even when in childhood, there is a slight stenosis of the orifice, this condition disappears naturally with growth and suitable treatment, it is always wise to wait until complications arise, and to make sure that these depend on the condition of the prepuce before interfering.

Of these two views the latter certainly appears to be the most sensible.

It is a safe rule, therefore, that all conditions of phymosis, in which retraction and reposition are difficult or impossible, and which give rise to symptoms, should be subjected to operation. The operation, whatever its nature, should be thorough, and lead to a sufficient and permanent widening of the prepucial orifice, with complete separation of adhesions, partial operations, such as insufficient dorsal incision, frequently causing increased phymosis through cicatrisation. The form of operation to be preferred varies, depending upon which variety of phymosis is present.

A. In the atrophic form, when the prepuce is stretched over the glans, and the outer layer does not project, a dorsal incision is usually sufficient.

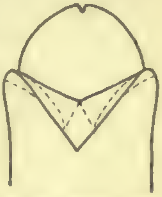
The patient having been prepared, a general anæsthetic is administered, though local anæsthesia, such as that produced by the local injection of cocaine, may in exceptional cases be employed. In cases where masturbation has been practised, it is recommended by some that no anæsthetic should be used, the moral effect of the pain being supposed to have a deterrent influence.

In youths and adults a red rubber catheter should be tied round the root of the penis to prevent hæmorrhage; in infants this is inconvenient and unnecessary.

Elaborate disinfection of the surrounding parts may be dispensed with, the penis being drawn through a small hole in the centre of a piece of lint, sterilised or moistened with warm, one in forty, carbolic solution, and the surrounding parts covered with sterilised towels.

The penis being fixed, and the foreskin firmly retracted with the left hand, a grooved director is carefully introduced between the prepuce and glans on the dorsal aspect, and along this the blunt blade of a small pair of scissors is passed as far back as possible, and the prepuce divided exactly in the middle line. The edges at once gape, so as to form an almost transverse incision, the inner layer being divided half-way up, the outer layer somewhat higher. By further traction on the skin of the penis, the outer layer is drawn back as far as possible, and from the angle of the inner layer a small triangular flap is cut with its base at the corona, and its edges meeting at an angle of 60°, its length being about half an inch in the adult and half that in infants. Irregularities of the edges of the inner layer are removed with scissors, and the angular extremities of the first cut rounded off. Any bleeding points having been twisted or ligatured, all adhesions between the prepuce and glans are separated, slight ones by means of a blunt probe, those that are firmer are cleared all round and then divided close to the glans, so as to wound the inner layer of the prepuce as little as possible, and thus prevent œdema; if necessary, any larger wound of the inner layer is closed with a fine catgut suture.

The interior of the prepuce is then cleansed with a one to forty carbolic lotion to remove smegma, and the wounds sutured. This is best done with a continuous suture of fine catgut so as not to require subsequent removal. One commences by adjusting the apex of the little triangular flap of the inner layer into the angle of the wound in the outer layer. This method is recommended by Roser as forming a skin edge at the apex of the incision, and thus preventing the rehealing of the original wound, with the formation of a firm, immovable cicatrix, or even a recurrence of the phymosis.


Roser's Modification.

Local after-treatment consists in keeping the part as much at rest and as aseptic as possible. A good method consists of dusting on a little powdered crystallised iodoform, and surrounding the part with a narrow strip of several layers of iodoform gauze, while over this a narrow carbolic gauze bandage is applied with moderate firmness up to the root of the penis, this acting as a kind of splint. Care must be taken to prevent the dressing as far as possible from becoming soiled with urine. It may be left on for several days unless the part be painful, being occasionally moistened with weak carbolic oil if it becomes wet. Another method recommended is to keep the part covered with a small strip of lint, kept moistened for the first twenty-four hours with a mild antiseptic lotion; this is then renewed and allowed to dry on.

Complete primary union is rare in children, but not uncommon in adults. Any point that remains unhealed is best treated by being smeared with a little soft boracic ointment.

The patient should be kept in bed for two days, and then allowed to lie on a sofa, but he is not to move about for a week. Bromide of potash may be required in adults if erections be troublesome.

B. In the case of hypertrophic phymosis which occurs so frequently in children, dorsal incision is unsatisfactory, leading to œdema and an inelegant result. Circumcision is, therefore, to be preferred. This operation, although it has undergone innumerable modifications, differs little in essentials from that originally performed and still practised as a religious rite by the Jews.

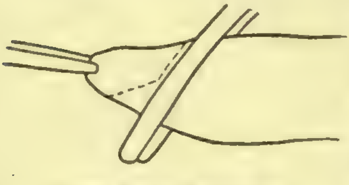
A point on the foreskin about a quarter of an inch in front of the corona is grasped obliquely from above downwards, and from behind forwards with long dressing forceps, or other special forceps, the obliquity making the resulting opening elliptical, and thus larger. Care must be taken that the glans is pushed back and is not included in the grip of the forceps. The anterior portion of the prepuce in the grasp of the forceps is then cut off by one sweep of a sharp bistoury.

The outer layer retracts to a considerable extent, and care is, therefore, necessary not to include too much in the forceps, otherwise the limb of the penis is apt to be left devoid of skin, this leading to delay in healing and subsequent discomforts during erections.

The inner layer still remains more or less enclosing the glans; it is to be divided by means of scissors in the mid-dorsal line almost to the level of the corona, or Roser's triangular flap may again be employed with advantage. The angles are then trimmed off up to the frenum, leaving a small fringe of mucous membrane surrounding the corona. Bleeding having been carefully arrested, the edges are brought together by means of a continuous or interrupted fine catgut suture, the former being preferable, but in the case of infants sutures are frequently unnecessary. If the triangular flap has been employed, it is well for exactness of adjustment to make a small mid-dorsal snip in the outer layer to receive it. The after-treatment is as formerly described. Should the wound not heal by first intention, as is usually the case at the frenum, warm baths and a mild antiseptic ointment are grateful and promote healing.

While these are the general principles upon which the operations should be conducted, there are, however, certain points to which further attention must be directed.

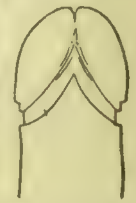
First, As to the employment of the elastic band to prevent hæmorrhage. When this is used, care must be taken before application to mark on the skin the exact position of the corona, otherwise one is apt to remove either too much or too little of the prepuce.



Davies Colley's Modification.

Second, The forceps having been applied, it is usual to cut behind them. Davies Colley, however, strongly recommends that the incision should be made in front of them, and the distal portion of the prepuce having been made tense, that the lower part of the incision should be made obliquely from behind forwards, so as to form a triangular flap of the middle of the under surface of the prepuce, which, he says, fits more accurately into the triangular gap with its apex at the stump of the frenum, which is formed on removal of the inner layer.

Third, When an elastic band has been employed in adults, primary union is frequently obtained and time saved by tying any obvious vessel, suturing and applying the dressing and bandage with moderate pressure before removal of the band. The subsequent swelling prevents further bleeding, but experience is necessary, as a too tight application of the bandage before removal of the elastic band may cause such subsequent pressure as may lead to slight sloughing of the edges of the wound.



Lastly, As to the question of operation in the case of gonorrhœa and inflamed hard, soft, and phagedenic chancres, the fear of infection and inflammation of the whole wound has frequently prevented this from being done when otherwise advisable; this, however, rarely occurs if the raw surfaces are well washed with carbolic lotion, any ulcerated surface not capable of removal being first dried and then touched with fuming nitric acid and iodoform rubbed in. In the case of great swelling and inflammation, a dorsal incision followed by disinfection and subsequent removal of the redundant flaps by means of scissors, no sutures being employed, often gives a better result than the more classical operation.

Circumcision is usually devoid of risk, but fatal cases have been recorded from cellulitis, erysipelas, tetanus, and hæmorrhage; while infection from syphilis and tubercle are not unknown as the result of ritual circumcision among the Jews.

LITERATURE.—1. Davies Colley. "Circumcision," *Guy's Hospital Reports*, 1893.—2. Günther. *Operationslehre.*—3. Jacobsen. *The Diseases of the Male Organs of Generation* and *The Operations of Surgery.*—4. Kaufmann. "Verletzungen und Krankheiten der männlichen Harnröhre und der Penis," *Deutsche Chirurgie*, Lieferung 50 a.—5. Saint-Germain. "Circumcision," *Nouveau dictionnaire de médecine et de chirurgie pratiques*, tome vii.—6. Treves. *A Manual of Surgery.*

Circulatory System.—General index of the main articles dealing with the circulatory system. Consult also index on "Blood."

Abdominal Aneurism.
Angina Pectoris.
Aorta, Thoracic, Aneurism of.
Aneurism (Surgical).
Arteries, Injuries of.
Capillaries, Affections of.
Electrolysis.
Heart—
  Physiology, Clinical Investigation of, Endocarditis, Chronic Valvular Diseases.
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  Neuroses of.
  Congenital Diseases.
Lymphatic System
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Pulse.
Vessels, Diseases of.
  (a) Arteries.
  (b) Veins.

Civil Incapacity—Capacity (Mental) for Civil Contract, etc.—It may be stated generally that a person while insane is legally incapable of entering into a contract; he is in the legal sense incapable of giving the consent which is essential thereto. Consequently he cannot enter into the contract of marriage, nor can he execute a will. Moreover, the management of his property may be removed entirely from his control.

What constitutes Legal Insanity.—It is not possible to formulate any criterion which would be generally applicable to the varied cases which arise. To generalise in the matter would be to mislead. The question of insanity in such cases is always the question of capacity intelligently to do the particular act. The late Lord President Inglis (in the Scottish case of Morrison, 1862, 24 D. at p. 631) said to the jury: "I am not going to give you any definition of insanity, and I am not even going to define to you what legal capacity is in a question of this kind, because I may tell you at once that the question whether a man is in such a state of mind as to be capable of executing a deed of this kind is a question of fact, and not of law. . . . The test of his capacity to execute such a settlement may be very reasonably stated with reference to the nature of the settlement itself, but it cannot possibly be stated without reference to the settlement, because a man may have strength of mind—power of intellect sufficient to enable him to do one thing, to make one kind of mental exertion—and yet he may be totally incapable of making another. A man may be so far weakened in his mind as not to be able to follow a difficult process of reasoning . . . and yet be perfectly capable of making a simple destination of his property. The two things are totally different. You can easily understand that a man may not be capable of very long-sustained mental exertion, and yet may be quite capable, reasonably and fairly, of saying, I want my estate to go to A. B., or I want to disinherit my heir and to leave my estate to an hospital. That is not a very complex idea; and if a person is capable of distinctly understanding what it is that he is doing—is capable of expressing that purpose in intelligible language, and is capable of understanding the consequences and effects of what he does—then he is capable of making such a settlement, and it is vain to go about scientific definitions or anything of the kind" (see also Erskine, J., in *Harwood*, 1840, 3 Moo. P. C. 282).

A deed disclosing no trace of incapacity or insanity may nevertheless spring from an insane belief or delusion; but in establishing this to the effect of invalidating the deed, it is necessary to prove not merely the groundlessness of the belief, but also the impossibility of its being entertained by any sane mind. The existence of a disordered belief upon some extraneous point, having no connection with the subject matter of a settlement, is not necessarily fatal to the deed. In a recent case the House of Lords (reversing a judgment of the Scottish Courts) held that these alleged facts, if established, would be sufficient to invalidate, on the ground of mental incapacity, the will of a testator who directed the greater part of his large estate to be employed in promoting total abstinence and preventing the spread of Roman Catholicism. It was stated that upon these topics he was subject to insane delusions, and believed that he had an imperative duty to devote his pecuniary resources to these objects in consequence of commands which he had received from the Deity by direct communication upon various occasions; and these insane delusions dominated his mind and mastered his judgment to such an extent as to render him incapable of making a reasonable and proper settlement of his estate (*Hope*, 1898, 1 Fraser, H. L. 1; see also Lord Penzance in *Smith*, 1867, L. R. 1 P. & D. 398).

It is necessary, then, that a testator should have sufficient memory to recall the amount and character of the property of which he is possessed, and to remember the several persons who may be said to have claims on him. Along with this memory, he must also have his judgment reasonably clear and free from morbid and insane ideas, so as to be able to decide as to these claims.

The question of the mental capacity of a person to enter into a particular contract or to execute a particular will is frequently combined with allegations of undue influence exercised by those who benefit by his act. The inquiry is then, of course, pointedly directed to determining whether he was facile, unduly liable to persuasion, easily incited to wrong views and feelings,—how far incapable of resisting pressure.

This incapacity to enter into civil contracts, etc., exists only when and *so long as* the person is insane. If he recovers his sanity permanently, or even temporarily, his contracts and deeds are valid. A will executed in a lucid interval, even by a person then confined in an asylum, is perfectly valid (see Nisbet's *Trs.* 1871, p. 9; Macpherson's *Reports*, p. 937). In such a case the rationality of the document itself is regarded as an important piece of evidence in favour of sanity. The burden of proof is shifted—that is all. Whereas a man is presumed sane until he has been proved to be insane, a person once proved to be insane is presumed to be insane until his recovery is proved.

Moral Insanity.—It has been judicially laid down in England (by Sir H. J. Fust in *Frere*, 1846, 1 Rob. E. R. pp. 442, 446) that moral insanity, unaccompanied by delusion, does not invalidate a will. But this can scarcely be said to be recognised in modern practice. There can be little doubt that proof of the insane perversion of the moral feelings would at least be held sufficient to invalidate a will (see Wood Renton on *Lunacy*, p. 55).

LITERATURE.—1. MAUDSLEY. *Responsibility in Mental Disease*, pp. 111 *et seq.*—2. RAE'S *Medical Jurisprudence of Insanity*, p. 2.—3. TAYLOR'S *Medical Jurisprudence*, ii. p. 545.—4. WOOD RENTON on *Lunacy*, pp. 8 *et seq.*, where cases are ably summarised.

Clavicle. *See* FRACTURES, SHOULDER-JOINT, STERNO-CLAVICULAR JOINT.

Cleft Palate. *See* PALATE.

Climacteric Insanity.

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THE NEUROSES incident to the change of life have not been studied with that care and accuracy which the subject demands. Although it is an everyday experience in medical practice to be called upon to undertake the treatment of maladies, more or less severe and distressing, complicating the menopause, the literature of the subject is singularly scanty. Dr. Tilt (first in 1853) published a book on *The Change of Life in Health and Disease*, which ran through several editions, and still remains the only English work of reference. His observations still rank as authoritative, although his explanations of facts must be regarded as obsolete. Various studies of affections of the menopause have been made, *e.g.* Dr. Saundby (1885) published certain observations on numbness of the extremities occurring at the climacteric, quoting Dr. Ormond and Dr. Wharton Sinkler. Dr. Barnes (1890) and Dr. Savage (1893) have written of the neuroses of the menopause, and Dr. Merson (*West Riding Asylum Reports*, 1876) has given an elaborate summary of the cases of climacteric insanity in that institution during the four years prior to publication. These communications represent a comparatively small volume of work in relation to an important epoch in the life-history of woman-kind.

It may be considered doubtful if there is a real analogous condition in men. The momentous epoch in a woman's life, when the menstrual function ceases and her general physical condition undergoes a profound alteration, is so marked and is so determined by physiological changes that it cannot fail to be taken into account, even in the most elementary consideration of epochal modifications. At puberty the child becomes a woman in form and feeling. The menstrual function is established and continues in periodic activity throughout sexual life. Any premature cessation of that function is accompanied by symptoms of ill-health, requiring active medical or surgical treatment; and when, in the course of vital involution, the function is being finally obliterated, it cannot but be, especially in the complex circumstances of the artificial life of civilisation, a period of stress more or less severe. The sexual life of a man, although, no doubt, obscurely periodic in character, has no such definite characteristics. The climacteric is but feebly indicated in the process of sexual decadence. No such active revolution, intimate in its connection with mind and body, forces itself upon medical attention. The familiar facts of the gradual slowing of life's current in men are ushered in by no such profound alterations as caused Madame de Deffand to exclaim, "Autrefois quand j'étais femme." For in this relation there is no question of premature senility, of organic degenerative nervous changes. The woman who has passed her grand climacteric is in no way enfeebled, it may be indeed that her mental powers are increased and that her physical energies are reinforced since the menstrual flux has ceased to be a recurring occasion of exhaustive and monthly disturbance. Still, when we consider the neurotic concomitants of the menopause in women, and find that these are usually marked by feelings of a melancholic type, a conscious loss of power, irritability, and general malaise, it must be admitted that similar nervous conditions do occur in men, although the period of life is more advanced, the symptoms are more fortuitous, and recovery is less frequent.

We have the high authority of Dr. R. Barnes in stating that probably few women go through the reproductive era without some nervous disorder. They labour under painful, dangerous tension, often concealing their distress, although sometimes on the verge of breaking down.

What, then, are the symptoms of the climacteric in women? First, there is a period of unrest, of flushing, giddiness, tinnitus, headaches, and general instability. In addition to subjective signs there are frequently floodings, or other uterine disturbance, which may pass on to organic diseases of the worst type, or may pass off and leave the person to the enjoyment of many years of excellent health. Too often these complaints are treated lightly, as part of the ordinary and inevitable routine of sexual decadence. Too often suffering is endured in silence and protracted by unnecessary reticence. But the physician is not warranted in assuming that these minor troubles preliminary to the cessation of menstruation are of slight importance and undeserving of active treatment. He can never be sure that nervous instability will not proceed to pronounced mental disorder, that floodings will not prove symptomatic of cancerous growths. Investigation and rational treatment must go hand in hand. Promptitude is essential.

Of late years the gynæcologist has been frequently forced to create an artificial menopause, and much blame has been deservedly cast upon those who have been held guilty of carrying operative measures to extremes. They have been charged, in unseemly terms, with needlessly unsexing women. They have been accused of creating greater evils than they removed. Cases have been recorded where the removal of ovaries has been detrimental to mental health, and the last state of the patient rendered worse than the first. An artificial menopause has been followed by a climacteric insanity.

In the earlier stages of neurotic maladies associated with the climacteric, the bromides are in common use. I have, however, found general and considerable benefit from the administration of ovarian substance in the form of tabloids. There is a disturbance of the animal economy consequent on excess of natural secretions, but there is also a disturbance dependent upon deprivation. If the sudden cessation of ovarian activity be accompanied by such symptoms as have been indicated, it is reasonable to suppose that the artificial ingestion of ovarian substance will in measure restore the balance which has been destroyed, and gradually accommodate the system to the new order of things which is imminent. It would seem that ovarian substance is in these slighter forms of nervous disturbance, at all events, a valuable remedy, to be prescribed on reasonable grounds.

Within the last few years, especially in America, few but active workers in asylums have pressed upon the attention of the medical profession their conviction of the urgent need for a more systematic, if not an universal, examination of the reproductive organs of insane women. Operative interference has been advised in these cases, and one enthusiast has placed on record that out of 100 insane women pelvic disease existed in 93, and operative interference was required in 89. Such a statement is altogether exceptional, and has been strenuously controverted. Cases have been recorded showing that the removal of ovaries has been followed by disastrous consequences, and that these operations, generally speaking, have as little effect in the cure of insanity as the discredited operation of clitoridectomy for epilepsy. Premature cessation of function, consequent on disease or consequent on operation, an artificial climacteric, is assuredly not less hazardous than the ordinary involution of middle age.

It is well known that certain bodily diseases are accompanied by certain mental concomitants. *Spes phthisica* is familiar to every physician, and the mental depression of liver disease has passed into a proverb. The influence of disorders of the sexual organs on mental states has been widely recognised. They may determine the production of hallucinations. For instance, it has been accepted that olfactory hallucinations are generally indicative of these disorders, and not less at the menopause. Ever since Schrœder van der Kolk insisted on the reality of *sympathetic insanity*, there has been no danger of overlooking the condition of the colon in the treatment of the insane; and perhaps more than enough has been made of the relations between the sexual and the mental apparatus. Indeed, in the active search made for underlying physical causes of insanity, the hopes which modern gynæcological treatment raised have been but partially realised. The correction of faulty conditions, however, offers a chance of mental recovery, and must not be neglected.

But it must be admitted, it must be kept in mind that the great cause of insanity is some inherited defect. Broadly, the neurotic inheritance is the foundation fact of mental disease. The stress of the climacteric period may tell severely even upon the strongest women. When that stress occurs in the case of a woman whose ancestry bears traces of mental weakness, whose mental instability is part and parcel of her very nature, the symptoms of earlier and less pressing importance, such as flushings and giddiness, should be regarded as indicative of a near possibility of further deterioration, mental and physical. The physician will seek to obviate these discomforts and disorders without loss of time, so as to prevent the development of morbid sensations into insane delusions, in so far as he can, by the regulation of vital functions and by the details of rational therapeutics and necessary hygiene. He will regard the stress of the climacteric as a presumptive exciting cause of mental disorder in a person constitutionally predisposed to nervous instability.

At such a time a woman may very readily slip into habits of invalidism. The nervous irritability, the physical weakness, the floodings may combine to induce her to adopt a course of life which is definitely detrimental. It may be that alcoholism, offering a speedy specious relief, is an immediate danger, although it is not so common a pitfall as has been recently alleged.

Alcoholic indulgence is not an efflorescence of late middle life; nor is it reasonable to suppose that the indecisive, introspective attitude so ordinarily adopted, intermingled as it is with religiosity and feelings of unworthiness, can be a favourable soil for vicious indulgence.

The question before the physician is one of redress. Equilibrium has been deranged. The habit of a generation has suddenly been broken; the system is not yet accustomed to the change of current. Manifestly his efforts must be towards the readjustment of equilibrium. This is not a mere question of medication of operative interference unless these are demanded by the very circumstances. It is rather a question of wise management of daily life, of diet, of exercise, of repose, of mental occupation and recreation than invigorating climate. To enter on the principles and details involved would mean a needless repetition of what will elsewhere be said in reference to the treatment of insanity.

INSANITY.—It would appear that the ordinary troubles of the climacteric pass into exaggerated forms when the boundary line of sanity has been passed. By fine gradations the natural feelings of advancing years, the weakening vital impulse, the questionings if work and pleasure still remain possible, pass into realised feelings of morbid dread, of physical incapacity, of unutterable unworthiness. The flushings are exaggerated into beliefs that something has given way in the head, for continuous headache is often referred to the vertex or to the occipital region. Feelings of emptiness may alternate with the belief that some live animal is crawling about the interior of the abdomen. The skin may be the seat of delusions. It is common to find patients complain of being filthy or diseased. The occurrence of obsessions to wash is noticeable at this period of life, as it often is about the time of puberty. Naturally the genital organs participate in these disorders, or even predominate. The belief that pregnancy has occurred is very common, although this condition of *pseudo-cyesis* is by no means confined to the climacteric period, nor is it necessarily insane. Every practitioner of experience will recall cases in which insistence on pregnancy at the climacteric has caused consternation and very real trouble. Sensations have been misinterpreted, and the cessation of the menses has been held as proof that the false interpretation of sensations has positive grounds of truth.

The varieties of insane delusions are numerous, and are developed out of these morbid feelings. As a rule, they have a sexual foundation. For instance, it is a common phase to complain of rape having been committed or having been attempted, especially in the hours of night. Not only may these patients complain of having been themselves criminally assaulted, but they may circumstantially relate that they have been made aware of friends or neighbours having been similarly maltreated. This opens up questions of medico-legal importance, which the practitioner will do well to bear in mind. He must exercise great caution in his dealings with persons so afflicted. And more especially is this to be remembered when they fall victims to epilepsy or epileptoid states, during which attacks there may be a performance of automatic actions requiring the closest observation in determining the responsibility of the parties.

The condition of life as regards marriage also determines the form of the mental malady. The old maid whose life has been narrow, and whose sympathies may have been warped, will probably suffer from an access of passionate feeling, the expiring flicker of the candle, and pester some unfortunate man with suggestions of marital relations, or accuse him of working upon her by means of electric batteries, or blowing chloroform through the key-hole in pursuit of nefarious designs. On the other hand, the married woman gives way to irritability, or dread, or disappointment, feeling that a new relationship has been established between her husband and herself. Instead of passing from the earlier years of married life to the years of a matron's duties, and so to a kindly green old age by natural and easy transitions, the patient becomes exacting and querulous, expends her energies in a passion of jealousy, and destroys the home which she had built up with fond care. These unfortunate first steps require a world of patience and most considerate treatment. They indicate a self-consciousness and a loss of control which, unchecked, pass to easily recognisable forms of mental aberration.

THE CLINICAL FORMS of climacteric insanity are broadly resolved into melancholia and mania, with comparatively few cases of delusional insanity of a melancholic type.

Melancholia is undoubtedly by far the most common form. The cares of life, the organic malaise of the climacteric ordinarily tinge the mental state with feelings of dread, fears of impending disasters, fears of impending disabilities. The consideration of the clinical features of melancholia need not be entered upon here. It is sufficient to correlate the depression and delusions with the underlying physical conditions.

Mania is less common, and would be still less common were it not that alcoholism plays a certain part in the development of maniacal conditions at the climacteric. Cases occur in which melancholia follows on alcoholic excess, but the usual concomitant is mania. Again, there are cases in which there is a recurrence of mental disorder at the climacteric. These would appear to be chiefly maniacal in form.

As to the cases of delusional insanity, where the predominant features are states of fixed and limited delusions, these are but few in number, and the delusions are almost invariably of a melancholic character.

Still fewer degenerate into dementia, and these are usually complicated by alcoholic or other toxic influences.

Dr. Savage has drawn attention to the observation of Dr. Tilt that deafness occasionally occurs at the menopause—a defect which may be temporary or permanent. No doubt tinnitus is frequently a symptom of the climacteric, and it may pass on to more serious conditions; but it does not appear that deafness is in any way peculiar in its mental complications at that particular period of life. It is a matter of common observation that deaf persons often become suspicious, morose, delusional, and dangerous. Those afflicted with congenital or acquired deafness and insanity are among the most dangerous of the inmates of asylums. They are nearly always persecuted and persecutors.

In addition to the mental aberrations above described and grouped according to preponderating symptoms, allusion must be made to certain moral aberrations which come under the notice of the physician from time to time. Reference has been made to the alcoholic habit which may begin with the troubles of the menopause, but which is more probably only a shameless exacerbation at that period. The excuse comes so readily that it gives the impression of having been too easily produced, just as the excuse may have been dysmenorrhœa or puerperal troubles in earlier life. It is not usual to find that a woman of alcoholic habits gives up her vicious indulgence on the cessation of climacteric disorders; on the contrary, it would appear that she continues to get drunk becauses she dislikes being sober. The deep-seated untruthfulness of the drunkard must be discounted in arriving at any opinion on this question. And, similarly, it is not in accordance with modern experience to expect that mental disease of long standing will cease and determine on the completion of the menopause.

More serious, if possible, are those cases of moral insanity in which nymphomania becomes a prominent symptom. Fortunately these are rare, but when they do occur, they are only too easily recognised.

Prognosis.—Having thus examined the general facts relative to mental disorders occurring at the climacteric, it is evident that there are no marked peculiarities other than can be readily understood on consideration of the underlying physical and environmental conditions. It will consequently be granted that on the cessation of the period of stress there is expectation that the recoveries will not be fewer than in connection with other similar disorders. Indeed, it may be averred that the chances of recovery are more than usually favourable, if we exclude cases complicated by previous attack, organic cerebral lesions, and the manifestly degenerate. Still recovery may be protracted, though but few die. The risk of death by suicide is comparatively small, notwithstanding the prevalence of gloomy thoughts and feelings of unworthiness, for the slower current of life enfeebles the power to end misery by self-destruction. Yet while this is generally true, it would be a reckless procedure which would fail to take such a risk into account and fail to take precautions for prevention.

The treatment of climacteric insanity must proceed on the theory of individual necessities. If the sudden cessation of ovarian activity is causing the mental instability, as has been already indicated, ovarian substance promises good results. If the symptoms are referable to the skin, and a harsh dryness indicates sluggishness of action, baths and exercise will be indicated. There is no class of cases for whom exercise is more generally beneficial, but exhaustion may be so profound that rest and massage are preferable. Or if the alterations in the appendages of the skin are the basis of delusions, these must be treated. Removal of unsightly hair from the face has led to recovery. Again, there is often constipation of an intractable form, leading to autotoxic effects. There is no better remedy for this than sulphate of magnesia in small and daily doses. These indications of treatment will serve to show how closely the physical condition must be studied, and how treatment must be appropriate to that condition. It may be that the gynæcologist will be required to operate upon new growths or to rectify malpositions. In short, if the treatment of insanity is to be successful, there must be no omissions in research and no failure in performance. This is not the place to enlarge upon those measures of mental hygiene which such patients require, nor is there any special note of interest in reference to the place of treatment. If appropriate treatment can be had without having recourse to asylums, no doubt that will be tried; but it will be recognised that, for the vast majority of cases of pronounced climacteric insanity, the special appliances and practical experience at the service of the public in a modern asylum are both requisite and necessary.

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Climate and Acclimatisation.

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Climate, from the Greek word κλίνω, I incline. Lat. *Clima;* Gr. κλιμα, a region; Fr. *Climat;* Ger. *Erdstrich*, *Himmelstrich*.

The word climate is now used to specify the definite differences which obtain between one region and another, as regards temperature and its variations, humidity and rainfall, the composition of the atmosphere, its density and pressure, winds, electrical conditions of the atmosphere, the organic and inorganic substances it contains, etc. The configuration of the ground must also be taken into account, the composition of the soil and the presence or absence of vegetation, marshland or desert, plain or valley, inland or maritime position, mountainous or otherwise.

The term used to be employed in astronomical or mathematical geography to designate "a portion or zone of the earth's surface, comprised between two lines parallel to the equator, and measured by the length of time during which the sun there appears during the summer solstice, that is, by the sun's inclination. The space between the equator and the pole was divided into half-hour climates, in which the length of each day increased by half an hour, and also into monthly climates."

As the climate of a region may be said to be essentially dependent upon the duration of its exposure to the sun's rays, modified certainly by local conditions, the relation of the region to the equator is of great importance. Hence, originally, three great climatic divisions were described—

1. The hot or warm climate, extending from the equator to lat. 35°, with a mean annual temperature of about 80° F. (27° C.).

2. The temperate climate, extending from the 35th to the 55th degrees of latitude, with a mean annual temperature of about 60° F. (16° C.).

3. The cold climate, between 55° lat. N. and the poles. In this region the temperature varies from 40° F. (5° C.) to 5° F. (−15° C.).

A more scientific division of climates is made by using isothermal lines, because the mean annual temperature varies in different regions occupying the same latitude on the earth's surface. This would give us five fairly extensive regions between the equator and either pole—

1. The hot region, between the equator and the isothermic line of 77° F. (25° C.).

2. The warm region, between the isothermic lines 77° F. (25° C.) and 59° F. (15° C.).

3. The temperate region, between the isothermic lines 59° F. (15° C.) and 41° F. (5° C.).

4. The cold region, between the isothermic lines 41° F. (5° C.) and 23° F. (−5° C.).

5. The polar region, between the isothermic lines 23° F. (−5° C.) and 5° F. (−15° C.).

Apart from temperature, rainfall, winds, etc., to which reference will be made presently, for practical purposes we must refer to seven climates—

1. Hot climate.
2. Temperate climate.
3. Cold climate.
4. Insular climate.

5. Maritime climate.
6. Continental climate.
7. Mountainous climate.

As all these climates have general differences, it will be well to briefly describe them before referring to details.

General Characteristics.—1. *The hot climate,* which may be described as practically extending to 35° north and south of the equator, contains within its limits South Asia, nearly all the islands of Polynesia, the greater part of Africa and its islands, and those parts of North and South America lying between the latitudes mentioned.

In these regions the heat is greatest, not, as might be imagined, at the equator, but at the tropics (20°-23° 30″). The heat at the equator is modified by the equatorial rains, which fall almost daily, and the equatorial calms, but although the rainfall is fairly equally distributed during the whole year it only amounts to about 45 inches in this equatorial region. Between 5° and 10° of latitude there are two rainy seasons in the year. In regions between 10° and 13° there is one rainy season, which lasts upon an average for five months. These general considerations are modified by the monsoons.

The variation in the thermometer is slight during the day, but at night there is often a considerable fall. In general terms, the range of temperature is from about 55° to 120° F. (13° to 49° C.). The daily variation in barometrical pressure is well marked, but the general variation is slight.

There is an almost rainless area north and south of the equator from about 16° to 28°, where it seldom or never rains, and in these belts the mean annual temperature is greatest. Throughout the hot climates thunderstorms are of frequent occurrence.

The general influence of the hot climate upon the inhabitants is to render them lazy and apathetic. It has a very definite influence upon the constitution of Europeans residing there, and the physiological functions of the body are distinctly affected. With regard to the normal *temperature* of the body, we find that, according to numerous observers, it is slightly raised, probably about 0°·40 F. (0°·04 C.), but the mean diurnal difference of temperature is practically the same in Europeans as seen in Europe. Crombie found that the mean temperature of Europeans in Bengal, taken in the mouth, was 98°·49, with a maximum daily range of 1°·31, as compared with the English average of 98°·084 and 1°·41 respectively. With regard to *respiration,* Rattray's observations are summarised by Davidson thus: "The vascularity of the adult lungs is reduced by 12 to 13 fl. oz.; their spiro-metric measurement by the average of 32 inches; their function by 18·43 per cent, that is, they use 36·85 cubic feet less of air daily; the excretion by 1·84 oz. less of carbon and 6·7 less of watery vapour." This is accounted for by the number of respirations being lessened to about 14 per minute, and there being considerably less blood in the lungs under the influence of an average temperature of from 80° to 83° F. (27°·28-5° C.).

There is a very slight difference in the *pulse rate,* and the proportion between the respirations and the pulse rate is not the same as in Europe. The amount of urine voided is diminished in quantity. Rattray says that it is diminished by 17½ per cent, but, of course, the amount of urine depends upon the amount of fluid consumed, the temperature, and the humidity of the atmosphere.

Opinions vary as to the effect of a hot climate upon the *blood,* and for this reason that probably malaria must be taken into consideration. On the whole, however, it is safe to say that Europeans suffer from a slight anæmia. By some authors this is considered physiological anæmia, and rather beneficial than otherwise.

The *nervous system* during the first few months of a residence in a hot climate is excited, but this excitement soon gives rise to depression, which latter is partly due to loss of sleep, partly to climate, and also to the worries incidental to a residence in a hot climate.

Digestion is slower than it is in Europe, and digestive powers are weakened; the appetite fails, and nutrition is diminished; weight is lost, and the muscular system enfeebled. The functions of the liver and skin are greatly stimulated. Menstruation commences about one or two years sooner than in Europe.

European children born in hot climates thrive fairly well in infancy, apart from their liability to suffer from diseases incidental to the climate.

The *mortality* in the hot climates in new arrivals is chiefly due to fever, heat apoplexy, and intestinal disease, but after a year or two's residence these diseases show a marked diminution in amount. It must also be admitted that sanitary measures and a more careful mode of living have greatly reduced the amount both of disease and mortality in Europeans in hot climates; thus the death-rate from disease in India among soldiers, excluding cholera, has decreased by two-thirds since the sixties, when the British soldier in Bengal died at the rate of some 60 per thousand. The death-rate of soldiers in Bengal in 1888, excluding cholera, was 10·54 per thousand. The mortality of the women, however, was somewhat higher. The death-rate of soldiers' children under one year was 189·64 in 1888, but it must be remembered that in India they do not suffer from the want, privation, and exposure to which children are subjected in the large towns of England, where the death-rate is very little lower, and in order to estimate these figures aright we must also remember that, as Davidson points out, "the diminished mortality is the result of withdrawing soldiers' children from the tropical influences, which proved so injurious, and rearing them in a temperate climate"; this is done by taking the children from the plains to establishments in the hills in the hot season.

2. *The Temperate Climate.*—This climate is the healthiest upon our globe, as neither the extremes of heat nor cold are experienced. It may be said to be situated between latitudes 35° and 55°. The mean annual temperature varies from 50° to 60° F. (10°-16° C.). In the temperate regions we have the four seasons—spring, summer, autumn, and winter,—and there are probably greater differences in local climates than elsewhere. It is in this area, too, that the influence of large towns and country districts must be taken into account, for, undoubtedly, the overcrowding which obtains in great cities does not tend either to longevity or to robust health, whilst infant mortality is very high. Great differences, however, obtain in different places, owing to the advance which sanitation has made. If we compare the death-rates of the various capitals of Europe we find marked differences; for instance, the death-rate in London is one-third less than in St. Petersburg or Moscow. Certainly the healthiest climates in the world are to be found in this temperate zone, and we possess far more accurate knowledge with regard to the diseases of this area than of any other. As will be seen in the article on "Health Resorts," they are practically all to be found in temperate regions.

3. *The Cold Climate.*—This climate, which covers the areas from about 55° F. (13° C.) to the pole, has been divided by some observers into three—the cold, having a mean annual temperature of from 40° to 50° F. (5°-10° C.),

the very cold, a mean annual temperature of 32° to 40° F. (0°-5° C.), and the glacial, where the mercury is below freezing-point. In the northern hemisphere, northern Russia, Lapland, Finland, Iceland, Norway, Sweden, Denmark, the north of Scotland, Spitzbergen and Nova Zembla, Canada, northern Asia, Siberia, and Kamtschatka, as well as Greenland, lie within it.

Although cold severely tries the lungs and kidneys, yet, according to M. Levy, the death-rate in this region is the lowest in the world. His estimate with regard to it may here be quoted:—

From	0°-20°	latitude,	1 death takes	place in	25	inhabitants.
„	20°-40°	„	„	„	35·5	„
„	40°-60°	„	„	„	43·2	„
„	60° 80°	„	„	„	55	„

The inhabitants of this region are vigorous and muscular, their powers of digestion are remarkable, but their nervous systems are not highly developed.

4. *The Insular Climate.*—This climate is remarkable in many ways; the seasons are more equable, owing to the surrounding water. The air is constantly changed by the prevalence of winds, and is charged with abundance of moisture.

5. *The Maritime Climate.*—This much resembles the insular climate. It is warmer in winter and cooler in summer than the continental climate, for, as a rule, the range of temperature increases from the coast towards the interior. The annual range of temperature on the coasts of the great oceans is diminished to about 20° F. (– 7° C.), whereas in the interior of a large continent, as, for instance, in the centre and north of Asia, the range may be from 60° to 100° F. (16°-38° C.)

6. *The Continental Climate.*—This climate has a tendency to extremes of temperature—cold winters, hot summers,—and even at a short distance from the sea these changes obtain.

7. *The Mountainous Climate.*—Here we have a low barometric pressure due to altitude, the air being more and more rarefied as we ascend. The heat diminishes, but the mountains attract clouds and watery vapour; this, notwithstanding, the climate is unusually healthy. The air is exceptionally pure. On first going to high altitudes, the respirations and pulse are accelerated, and the amount of carbonic acid and water exhaled by the lungs is increased, but after a residence of from one to four weeks, the pulse falls to normal, the respiration, however, continuing fuller than it was on the plains.

A consideration of the ocean climate will fall more naturally under the heading of "Health Resorts," but it may be said to be essentially characterised by warmth, equability, and excessive moisture.

INDIVIDUAL FACTORS.—Considering now more in detail, but yet briefly, the various factors which are summed up in the comprehensive term of climate, we turn first to the *composition of the air.* Roughly speaking, in 100 volumes of air, we find there are of oxygen 20·96, nitrogen 79·00, carbonic acid 0·04. The amount of oxygen in the air varies to a considerable extent, but rarely exceeds one-tenth in volume; the carbonic acid in the atmosphere varies considerably also; it may be in the proportion of from 4 to 30 in 10,000; there are also in the atmosphere other substances of a gaseous nature—ammonia, ozone, etc., as well as solid substances floating in the air, which vary in different places and under various conditions. Obviously in large towns one would expect these adventitious particles to be far more abundant than in the country. The suspended matters in the atmosphere are of considerable importance, and indeed it is upon the recognition of these that Lister's antiseptic theory has been based.

The *temperature of* the air naturally also influences climate, and in this connection we have to distinguish between the sun heat or radiant heat, and the air heat or shade temperature, which is due to the warmth imparted to the air from the ground, unless influenced by wind. "The nature of the surface of the ground exercises great influence on the amount of heat which is absorbed and reflected; the nearer the colour of the ground approaches to white (snow, chalk cliffs, white walls, etc.), the more direct the sun heat reflected by it, the less heat being absorbed; the darker the ground (grass, green leaves), the less heat reflected and the more absorbed. The ground which absorbs more heat from the direct rays of the sun can give out more heat during the night, and *vice versa.* The influence thus exercised on the climate of a place is evident, and the white snow-fields of the Alps in winter form a well-known illustration."

The temperature of maritime regions is influenced by the ocean, and in mountainous regions altitude lowers the mean annual range of temperature.

With regard to *barometric pressure,* it must be noticed that, if great, it exerts a very marked influence upon the human being. It tends to increase the amount of carbonic acid and water exhaled by the lungs, and at first to induce laboured respiration and inability for exercise, as well as constipation and depression.

The *influence of light* is also important, and, owing to its action on the centripetal nerves, it increases the metabolism.

Winds purify the air and induce changes in temperature, atmospheric pressure, moisture, and light, and therefore they must be taken into consideration, not only with regard to climate in general, but as to health resorts in particular. The monsoon winds, the sirocco, and the kamsin, are beneficial or untoward factors where they obtain.

The *cultivation of the soil* exerts an influence upon the temperature, for cultivated land, which is well drained, raises the temperature, as has been demonstrated by Buchan. It is obvious also that vegetation must play a marked part, because where it is luxuriant the sun's rays upon the ground are more or less prevented. Ground covered by forests has a lower temperature than that which is bare, and the diurnal variation in well wooded districts is more equable than that in open country. It is obvious also that sandy, dry, and well-drained soils are much warmer than wet and marshy districts, which certainly produce mist and fog. A sandy desert is exceptionally warm in summer, although comparatively cold in winter.

The amount of *watery vapour* in the atmosphere is also an important factor of climate. Evaporation and condensation constantly alter the humidity of the atmosphere. As a rule, absolute humidity is greatest with a high temperature, but relative humidity is usually greater in winter than in summer. It is only when saturation of the air obtains that we have mists and clouds,—clouds predominating at average heights, mists clinging to plains and maritime positions, or the neighbourhoods of large lakes. On mountain tops mists are rare.

With regard to *rainfall,* its distribution is unequal over the globe and varies in amount, from 300 or 400 inches annually on the southern slopes of the Himalayas, to almost nothing in the Sahara. Again, the number of rainy days experienced annually varies greatly and does not invariably correspond to the amount of rain which falls. Rainfall is not always injurious, as it diminishes the amount of impurity in the atmosphere; but, in considering the climate of a health resort, the amount of rain, the rainy season, the number of days on which rain falls, and the time of the day, are all subjects for consideration, as far as invalids are concerned.

It should be remembered that where mountainous regions lie in the path of moist air currents rainfall is great, as, for instance, the Khasi range of hills, which have an annual rainfall of about 500 or 600 inches, for they intercept the S.W. monsoon, which, laden with vapour from the Bay of Bengal, impinges on them. On the lee side of mountains, however, the rainfall is less.

Although the electrical conditions of the atmosphere must play some part in climatology, its practical importance is insufficiently understood.

Climate must also be considered with reference to the treatment of disease. A change of climate cannot be said to be a specific in treating disease, but, there is no doubt that in many diseases a change of climate acts most efficaciously. It is a well-known fact that the removal of a patient for even 20 or 30 miles may be exceedingly beneficial. In choosing a change of climate for an individual, the patient's personal equation or idiosyncrasy must be taken into account. In dealing with patients suffering from nervous diseases, in whom we wish to restore the general tone of the system, we should choose bracing and moderately temperate climates, remembering that proximity to the sea may induce nervous excitement and insomnia, and may, temporarily at least, exaggerate neuralgias. Diseases of the lungs, apart from phthisis, are favourably influenced by a climate characterised by moisture and mildness. A bracing equable climate is requisite in cardiac disorders, remembering that pulmonary complications due to climate should be avoided. A moderate altitude is usually beneficial, but the patient must not be sent too rapidly to a high altitude, and very sudden changes of temperature must be avoided. In diseases of the abdominal organs, such as intestinal catarrh, chronic liver disease, dyspepsia, dysentery, diabetes, etc., change of climate may be most beneficial; climates such as the Riviera, the Nile, St. Moritz, and Morocco, being advisable. This will be further considered under "Health Resorts."[1]

ACCLIMATISATION.—The question of acclimatisation naturally falls under the subject of climate. In recent years two schools of thought have been formed—one regarding acclimatisation as impossible, a view supported by Virchow, Hirsch, Fritsch, Ravenstein, and others, who hold that Europeans can never become acclimatised in the full sense of the word in tropical regions, believing that the deterioration caused by climatological factors and endemic diseases will invariably kill off the emigrants, or at any rate render their ultimate existence impossible; the other school, represented by De Quatrefages, Livingstone, the late Bishop Hannington, and more recently by Dr. L. Sambon, believing that rapid acclimatisation in tropical regions is possible for Europeans. The writer of this article holds that rapid acclimatisation in tropical regions is impossible, and that acclimatisation for Europeans can only be possible if migration occur step by step. In estimating the possibilities of acclimatisation we must count by generations rather than by years, although, given carefully selected individuals and carefully selected tropical areas in which to colonise, he sees no reason why, with precautions, Europeans should not colonise even in the tropics. In making the selection, all persons with a tendency to gout or rheumatism, diabetes or albuminuria, those with a nervous or alcoholic family history, or those suffering either from acquired or hereditary syphilis, should certainly be rejected.

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[1] See also "Hygiene, Tropical."

Acclimatisation is a process, usually slow, by which plants and animals become adapted to, and so retain health in, countries having a different climate from those in which they are indigenous; it is in part affected by changes taking place in the individual or in the race, in part by hereditary modification of constitution. If we look at the distribution of different races throughout the world, we find that great changes have taken place in their location; the Esquimaux once lived in Asia, only some 40° north of the equator; now they inhabit the polar regions; the Bohemians or Gipsies are found nearly everywhere, while Jews and Maltese apparently thrive in every conceivable region. But, examining the matter more closely, we find that the Esquimaux did not arrive at their present hunting-grounds in a generation, much less in a few weeks, as emigrants and soldiers are compelled to do now, nor do we find that the Gipsies or Jews overspread the world rapidly. Far from it; they put out feelers, as it were, and only very gradually, year after year, generation after generation, did they advance from their native soil into the great unknown. Looking for a moment at other races, we observe that the British become acclimatised at the Cape, in Southern Australia, in New Zealand, and in some parts of North America, but not in India. The French thrive in Nova Scotia and in Mauritius, but not so well in the north of Algeria, although in the southern provinces they do better; the Dutch fare exceedingly well at the Cape, but not in the Malay Peninsula, where they have experienced fearful mortality. Madagascar may be instanced as a place having a climate which has proved most unfavourable to all Europeans.

In dealing with the subject of acclimatisation with reference to Europeans, an error is made in imagining that all Europeans can be acclimatised or will resist acclimatisation in any given area equally; not so, a marked difference obtains between northern and southern Europeans in this respect. Not only the climatology of their original residence must be taken into account, but also their habits and customs and their psychical peculiarities, and if these factors have to be considered with regard to the adaptability of a nation for emigration, so too with regard to the individuals of any nation. Selected individuals from nearly every European nation may thrive almost all over the world. Some have denied that environment influences the human species, and it has been said that nations seek out that environment which is best suited for them; but environment certainly does definitely influence not only individuals but nations. Herbert Spencer has shown in his *Principles of Biology* that every organ and every function of living beings undergoes a certain and definite modification, within certain limits, under the stimulus of new conditions; and he thinks that this modification is almost always such as to produce an adaptation to a new environment.

The influence which climate and environment exert upon emigrants is well shown in Australia, New Zealand, and America, proving that a race such as the Anglo-Saxon may undergo material changes. It is obvious that the Colonials of the present day are practically different from their ancestors, and they indicate a tendency to change to an altogether new type of manhood, with new aptitudes and capabilities.

Broadly speaking, Europeans can only become rapidly and readily acclimatised in the temperate zone, that is to say, where climatic and other conditions are approximately akin to their present habitat. It may also be said that people who inhabit the temperate zone become more easily acclimatised in countries towards the north of their present habitation.

It is interesting to notice that the peoples of Southern Europe, such as the Italians and southern Frenchmen, can better bear the climate of subtropical Africa than can Northern Europeans, and, as Mr. Ravenstein has pointed out, "a steady stream of migration is, in fact, setting in that direction. Germans and Belgians are pouring into France, Frenchmen are going to Algeria, the Arabs from the shores of the Mediterranean have found their way into the Sudan, whilst the Sudanese are pushing forward into Bantu Africa. The descendants of those Dutchmen who, a couple of hundred years ago, first settled at the Cape, have made their way to the Transvaal, and European migration, favoured by geographical features, is being pushed, even within the Tropics, towards the Zambesi."

In treating the subject of acclimatisation, some have simply divided mankind into two classes, white and black, but this is a far too sweeping generalisation, and it must be examined more closely if a definite and right conclusion is to be arrived at. An endeavour must be made to prove with certainty in each separate case what power of resistance is possessed by any given national constitution, in order to decide whether it may successfully acclimatise itself in a new country and permanently colonise. In white races there is apparently a marked gradation in their susceptibility to climatic influences and a very decided difference is noticed between the Aryan and Semitic races in this respect. The Arabs and the Jews are both unequalled in their power of adapting themselves to new environment, but even between them a difference exists, in so far as the Jews appear to continue definitely capable of reproduction, although they marry amongst themselves, while the Arabs often suffer from degeneration which is only retarded by the introduction of new blood. They take wives from the races among whom they settle and thereby continue to exist, but this is not true acclimatisation, for the race is altered and transformed into a mixed race, which eventually possesses very little in common with the original stock. Race itself does not always provide us with a definite clue to capability of acclimatisation, for in India the Hindu population, notwithstanding its Aryan origin, has thriven under unfavourable circumstances and even in malarial districts, presenting a striking contrast to the English (also an Aryan race), whose intolerance of the Indian climate is obvious. Temperament also plays a not unimportant part in the possibility of prolonged existence in a given region. It is a quality which requires considerable time to be modified by new conditions of life, *e.g.* the Indian at Brazil is dull and sullen, the negro is vivacious and gay, although in the same climate. Hereditary temperament and capacity of mind characterise variously all races, and differences of intellectual and moral power are well-marked factors in the possibility of acclimatisation. Virchow not long ago pointed out, with reference to acclimatisation, that two distinct questions are usually confused, viz. (1) How long can any single individual, with precautions and care, live in any particular climate? (2) what races can thrive and colonise in any particular climate? No definite answers are yet possible. In answer to the first it may, however, be said that selected individuals may live for a time anywhere with precautions. With regard to the second, rapid acclimatisation of a race can only be obtained if it migrates to regions having approximately the same climatic conditions as its original habitat. If Europeans, for instance, attempt to colonise in the tropics, they almost invariably die out. In the West Indies we have a striking exemplification of this, and even in Cuba, which is brought forward by some authorities as a proof to the contrary, the apparent increase in the white population is more than accounted for by immigration. The permanent decrease of the white population in the West Indies dates from the abolition of slavery; as overseers and occupiers of the soil, Europeans are able to withstand climate more successfully than when they are compelled to undertake actual manual labour. The white man's incapacity for manual labour is an important question throughout the tropics; it has necessitated the employment of Chinese labour in America, of coolies in Mauritius and Java; and of kidnapped natives from the South Sea Islands in Australia.

From what has been said it is obvious that Europeans are *almost* incapable of colonising in the tropics—*almost*, because there are some places in the tropics occupying a high altitude, and some islands whose climate is so modified by the surrounding water and by their exposure to winds, that they almost possess a temperate climate. For instance, in the Vindhya hills in India the French have colonised successfully, and, theoretically, there is no reason why Europeans should not colonise and thrive in some of the highlands of Central Africa, when railway communication has been provided to carry them rapidly across the dangerous belt of malaria on the coast.

If we desire an example of a race thriving in extreme vicissitudes of climate, it is to be found in the Indians, who, coming from a temperate region in North America, have become acclimatised in the hot dry coasts of Peru, and also in the extremely cold regions of the Andes at an altitude of 4000 to 6000 ft. Now, notwithstanding that the inhabitants of these two regions will not thrive if they are removed from one to the other, they have nevertheless proved the possibility of an original race becoming gradually acclimatised in areas which are totally different from a climatological point of view.

Bertillon well summed up the circumstances which go far to prevent sudden acclimatisation in new isothermal regions:—

1. Acute diseases, many of them endemic.
2. Chronic consecutive anæmias, which place the individual in an unfavourable position to resist accidental disease.
3. Diseases in early infancy in offspring in the new home.
4. Physical and intellectual degeneration and the infertility of the second and third generations.

That climate markedly affects the progeny of emigrants is certain; for instance, when Europeans proceed to countries where they can colonise, which possess a very different climate from their own, their children in a generation or two receive an indubitable impress from the climatological factors around them. The children of Europeans who go to North America tend to approach in type to the original inhabitants of the soil; they outwardly resemble the north-west American Indians; the hair becomes straighter, coarser, darker, the cheek bones more prominent, and the rounded form of the face changes into the gaunt-lined face typical of the inhabitants of the United States; the skin becomes somewhat sallow, and even the voice changes. Again, supposing a woman proceeds to Australia, having given birth to three or four children; the children she subsequently bears in the new climate will not resemble her previous progeny so much as they do the Australian type. In tropical countries we find that the children of northern European families do not thrive, and the race dies out, not so much because of the infertility of the women, but because of their inability to bear children capable of thriving. In those cases where the women are sterile it is due to the action of the climate upon them as Europeans, and not to any specific action on the sex.

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Clitoris. *See* Vulva.

Club Hand and Foot. *See* Deformities.

Coal-Gas Poisoning. *See* Toxicology.

Cocaine.

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Cocaine is an alkaloid which was discovered by Niemann as long ago as 1860 in the leaves of *Erythroxylon coca*, a shrub cultivated chiefly in Bolivia and Peru. It was introduced into clinical use by Koller in 1884. In the British Pharmacopœia the official preparations are the dried leaves—Coca; extractum cocae liquidum; cocaina; cocainae hydrochloras; lamellæ cocainae, each of which contains $\frac{1}{200}$ of a grain of the hydrochlorate of cocaine; injectio cocainæ hypodermica; unguentum cocainæ; and trochisci krameriæ et cocainæ.

The Alkaloid.—Cocaine is but slightly soluble in water. It dissolves readily in alcohol, in melted vaseline, in castor and other fixed oils, and in many volatile oils. If heated in water it decomposes, and the solution contains ecgonine, benzoyl ecgonine, and benzoate of cocaine.

The Salts.—Hydrochlorate of cocaine, the officinal salt, is readily soluble in water, but insoluble in fats and oils.

Physiological Action.—*Local.*—When a solution of hydrochlorate of cocaine is applied to a mucous membrane it produces temporary local anæsthesia and anæmia, which last for ten minutes or more, according to the strength of the solution used. If applied to a turgescent mucous membrane, it produces collapse of the swollen structures, unless the turgescence has been so long continued as to result in connective tissue hypertrophy. Temporary local dilatation of the blood-vessels may follow.

When applied to the conjunctiva, slight smarting, followed by anæsthesia, results. The pupil is dilated, an effect ascribed by Koller to contraction of the vessels of the iris, but by most other authorities to irritation of the sympathetic. Accommodation is impaired, but this passes off more rapidly than the paralysis produced by atropine.

The skin is much less affected by local application than are the mucous membranes.

Internal.—Internally cocaine acts as a stimulant, producing a sensation of exhilaration and well-being, with increased mental and muscular power. In animals the brain, medulla, and spinal cord are stimulated from above downwards. Large doses produce convulsions of cerebral origin. The sensory tracts of the cord are paralysed, and anæsthesia results. This effect has been produced in animals by the administration of dangerous doses, but quite recently Bier has produced a similar effect in the human subject by the injection of small doses ($\frac{1}{6}$ to $\frac{1}{4}$ gr.) directly into the cerebro-spinal sac under conditions similar to those required by Quincke's exploratory puncture. He claims that complete anæsthesia can thus be safely produced below the level of the nipples. He has operated thus successfully on several patients chiefly for tuberculous bone and joint disease of the lower extremities.

Circulatory.—Cocaine produces moderate stimulation of the heart's action.

Respiratory.—Respiration is powerfully stimulated, and its rhythm is disturbed. Death from cocaine poisoning results from paralysis of respiration and the exhaustion produced by the accompanying convulsions.

Large doses produce a slight rise of temperature. Cocaine is mainly destroyed in the body, but may in part be eliminated by the kidney. The amount of urine is said to be slightly increased, and the urea to be diminished.

METHODS OF APPLICATION.—The great value of cocaine lies in its property of producing local anæsthesia, and for this purpose various methods of application are adopted.

For the *eye* aqueous solutions of the hydrochlorate of the strength of two per cent, or even of four per cent, are made use of, a drop or two of the weaker solution requiring to be applied two or three times at intervals of three or four minutes.

For the *mucous membranes* stronger solutions may be made use of even up to fifty per cent, but it is seldom necessary to use solutions stronger than five per cent. Several applications of the weaker solutions may be made at intervals of three or four minutes, or a small piece of cotton wool soaked in the solution may be left in contact with the part to be rendered anæsthetic.

For application to *the skin*, watery solutions are of little value, and for this purpose oily solutions of the alkaloid should be made use of.

Injected hypodermically, anæsthesia is produced for a limited area around the puncture. A watery solution is made use of, and the dose as a rule should be limited to half a grain. Where possible, *e.g.* in amputation of a finger, a ligature should be tied round the part to be operated on in such a way as to obviate the risk of toxic effects from the passage of the drug into the system.

Another important method of application is that known as the infiltration method (Schleich). This is described in article Anæsthesia, vol. i. p. 211.

USES.—*Local.*—The degree of anæsthesia produced by cocaine when applied to the eye is sufficient to allow of practically any operation being performed on the eyeball. It meets all the requirements that can reasonably be expected of a local anæsthetic, and hence has come to be regarded in ophthalmic practice as the anæsthetic *par excellence.*

In nasal, aural, and throat operations it is of great value, and the manner of application and other points of practical importance will be specially referred to under "Ear" and "Nose." In dentistry it is useful in toothache, and is also used to deaden the exposed pulp or dentine before filling the cavity of a carious tooth. By injecting a dose into the gum on each side of a tooth the pain of extraction is greatly lessened. By hypodermic injection or by Schleich's infiltration method nearly all minor surgical operations can be carried out painlessly.

During labour it has been used to relieve the pain of the dilating os uteri, and to relieve spasmodic contraction due to pain.

An oily solution of the alkaloid may be used to relieve the pain of shingles, eczema, neuralgia, pruritus, or urticaria.

Acute coryza, acute pharyngitis, hay fever, and similar conditions may be much relieved, especially in early stages, by a spray of a watery solution.

Internal.—Cocaine is used internally for sea sickness and the vomiting of pregnancy, as a tonic during convalescence, in mental exhaustion and muscular debility, and it has been recommended in alcoholism and the opium habit, but its use in the latter cases must be strongly condemned on account of the risk of development of the cocaine habit.

Drawbacks to the Use of Cocaine.—One of the greatest dangers attending the use of cocaine is the development of the cocaine habit. This habit is frequently secondary to morphinism, but may be developed from the medicinal use of the drug. In one of several cases which have come under the writer's notice, the habit developed in a chemist who was wont to resort to a cocaine snuff to relieve a chronic nasal catarrh. The "coca wines" so recklessly pressed upon the public as useful "tonics" are also a source of danger (see "Morphinomania").

When used with caution untoward effects are not common, but such have occasionally occurred, especially after the too free application of solutions to the nose and throat. Symptoms noted have been giddiness, faintness, pallor, feebleness of the pulse, great dyspnœa, and final collapse into unconsciousness.

It is difficult to keep solutions sterile, and it must be remembered that solutions cannot be boiled without destroying the cocaine.

Eucaine.—To obviate the disadvantages, and even dangers, occasionally attending the use of cocaine, efforts have been made to produce some substance free from these drawbacks. These have resulted in the introduction into practice of two synthetic products known as eucaine, both of extremely complex composition. For convenience these are distinguished as alpha-eucaine and beta-eucaine. The former of these was found to be less toxic than cocaine and to be an efficient anæsthetic, but, unfortunately, when applied to delicate mucous membranes it produced a great deal of smarting and irritation, and thus was unsuited for ophthalmological work. For beta-eucaine it is claimed that it is equal to cocaine in anæsthetic properties, that it is much less toxic, and that it does not cause local irritation. It is soluble in distilled water to the extent of 5 per cent, and the solution can be sterilised by boiling. Moreover the solutions keep well.

Beta-eucaine may be used for all the surgical procedures for which cocaine is adapted, and in solutions of from 2 to 10 per cent. The stronger solutions must be made with hot water. For ophthalmological work a 2 per cent solution is recommended. It differs from cocaine in that it produces no dilatation of the pupil, and no disturbance of accommodation. Another point of difference is that it causes little or no contraction of turgid mucous membranes, a point which may be an advantage or a disadvantage according to what is desired.

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Coccygodynia.—*Definition.*—A painful condition of the coccygeal region produced by the acts of sitting, walking, or defæcation.

Description.—The structures involved may be the coccyx, sacrococcygeal joints and ligaments, perineal muscles attached to the bone, terminal branches of the sacral plexus of nerves, and probably the coccygeal gland. It has to be distinguished from a somewhat similar condition frequently associated with certain affections of the vulva, uterus, ovaries, pelvic peritoneum, and separation of the bones at the symphysis pubes. It is found almost exclusively in the female sex, although cases are recorded of its occurrence in males and children.

Etiology.—1. Traumatism during labour, causing:—

(*a*) Injury to the fifth sacral and coccygeal nerves.

(*b*) Dislocation of the sacro-coccygeal joint, or fracture of the coccyx.

The latter may be produced by other causes, *e.g.* falls or blows.

2. Rheumatism affecting the ligaments or the periosteum of the coccyx.

3. Neuralgia affecting the terminal branches of the sacral plexus.

Probably most cases are of this nature.

Diagnosis should be made by placing one finger in the rectum and the other over the skin surface of the coccyx, when pain is produced on pressing the bone or structures round it, according to the part chiefly affected.

Symptoms.—Pain limited to the coccyx and neighbourhood, and aggravated by such acts as sitting or walking.

Prognosis is favourable, although many months may elapse before the pain completely subsides, and the condition is prone to recur.

Treatment.—Any concomitant pelvic disease must be first attended to. If there are indications of a primary inflammatory condition of the parts affected the patient should be kept at rest in bed, preferably in the lateral posture. Pain may be relieved by lead and opium fomentations, morphia hypodermically, or a suppository of morphia and belladonna.

Laxatives should be administered to facilitate defæcation, which is frequently painful. In cases associated with dislocation or ankylosis of the sacro-coccygeal joints, massage and manipulation should be tried before resorting to operative measures. If there is no lesion of the bone the faradic current gives excellent results, one pole being applied over the sacrum and the other over the coccyx. As a rule two to twelve applications are sufficient. The application of the actual cautery over the origin of the sacral nerve may be resorted to if other means fail to give relief. The cases recorded as occurring in men and children have been successfully treated by antirheumatic remedies.

Operative Treatment.—In obstinate cases Sir J. Y. Simpson recommended and carried out the subcutaneous division, by a tenotomy knife, of the muscular and fibrous tissues inserted into the sides and apex of the coccyx, with the object of preventing any movement of the bone. When the condition is associated with a lesion of the bone and unrelieved by other methods, Nott first suggested the complete extirpation of the bone. To carry this out a vertical incision is made over the posterior surface of the bone; the apex is then pulled well back, allowing the muscular attachments to be freed; finally, the bone is separated at the sacro-coccyginal point.

Both these latter methods are rarely necessary, and cases are recorded where the pain recurred even after operative treatment.

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Colic.—Under normal conditions the muscular coat of the digestive tract performs its functions of mixing and propelling food from the stomach downwards, and of finally expelling the fæces, painlessly; but, under numerous abnormal conditions, its gentle, painless, and harmonious working may be roused into violent, painful, and irregular action, the symptoms of which are designated by the term colic (from the Greek κῶλον). Colic, as ordinarily understood, may therefore be defined as abdominal pain due to spasmodic and painful contraction of the alimentary musculature. Other hollow tubes or hollow organs may be the seat of similar morbid action, and may exhibit like symptoms, and the original term has been extended to the gall-bladder and bile-ducts (hepatic colic), and to the kidney and ureter (renal colic). But this article will deal solely with colic as occurring in the musculature of the digestive tract.

Symptoms.—The essential and outstanding symptom is pain, located in the abdomen, remittent or intermittent, writhing or twisting in character, and generally relieved by pressure. Its onset may be sudden or preceded by premonitory uneasiness. It may be localised to a particular part of the abdomen, but if severe, its origin cannot be defined, and it is felt generally over the abdomen with its maximum around the umbilicus. It is spasmodic, coming in waves, shorter or longer in duration, and rising from the level of complete absence of suffering, or of more or less persistent uneasiness. It varies in severity from bearable griping to agonising spasm, and in duration from a few minutes to several days. As in all abdominal pain, the patient intuitively flexes the trunk and draws up the limbs, but, as pressure generally relieves rather than aggravates his suffering, he indulges the desire to move or roll about, at the same time applying pressure in various ways, such as rubbing by hand or leaning against a pillow. Where there is considerable bowel distension or associated inflammation pressure aggravates the suffering. The pain is of a peculiarly depressing character, the face is pale and indicative of suffering, the skin is cool and possibly moist, the pulse is normal, or weakened and slowed rather than quickened, the temperature is not raised, the bowels may be seen, felt, and heard to be in excited peristalsis, vomiting may or may not be present, the bowels may be obstinately confined, or enabled sooner or later to expel their gaseous or other contents with gratifying relief to the distressing symptoms. If the cause of the colic be towards the anus the pain is associated with a bearing down and expulsive desire. Vomited matters are at first from the stomach, but eventually, when the cause of the colic is bowel obstruction, they are from the bowels. The abdomen may be retracted or distended according to the amount and character of the bowel contents, and the condition of the intestinal and abdominal musculature. The breathing is interfered with according to the degree of associated contraction of the respiratory muscles.

Pathology. — The essence of colic is excessive contraction of the nonstriated fibres that form the alimentary musculature, and it has, as its analogue, the tetanic contraction of striated muscle that gives rise to cramp. The alimentary canal possesses within itself, independently of the central nervous system, the nervous mechanism necessary for peristalsis which can be directly stimulated. But the bowel movements are also influenced and secured by stimuli acting through the motor or augmentor vagus, reflexly or from the cerebral centres. Hence the abnormal or excessive stimuli that lead to colic may be local, as they generally are, or they may be distant, acting reflexly or directly from the cerebral centres. The pain is not a neuralgia in the ordinary sense of the term, but is caused by pressure on nerves from severe muscular contraction. As the splanchnic nerves inhibit peristalsis they can have no causative influence on colic.

The inclusion of visceral neuralgia under the term colic is undesirable.

Diagnosis.—The diagnosis of colic would be comparatively easy were it not frequently associated with ailments which complicate and obscure its manifestations. It must be diagnosed from other diseases, from other colics, and colic occurring in one part of the alimentary canal must be differentiated from colic in another.

1. From other diseases—

(*a*) *Inflammation.*—In inflammation the chief distinguishing symptoms are that the pain is increased on pressure, that it is more or less constant, and that fever is present as shown by quickened pulse, hot skin, and raised temperature.

(*b*) *Neuralgia.*—In neuralgia the pain is shooting or stabbing in character, tends to dart in various directions, to be increased rather than relieved by pressure, and to be associated with superficial hyperæsthetic areas. There is a history sufficient to account for lowered health, and there are present other signs of the neurotic temperament.

(*c*) *Angina Pectoris.*—Certain cases of angina pectoris are difficult to distinguish from abdominal colic, and have to be kept in view in making a diagnosis.

2. From other colics (Hepatic, Renal, Bladder, Uterine):—

(*d*) In *hepatic colic* the pain is in the upper part of the abdomen, and is towards the right side and right shoulder. Excited gastric or intestinal peristalsis is absent. There may be the history of previous attacks. After an attack there is tenderness on pressure over the gall-bladder. There may be jaundice, and, if gall-stones be expelled, they are to be found in the stools.

In *renal colic* the pain is in one or other flank; it shoots down towards the bladder and into the penis and testicle; it is associated with frequent desire to micturate, and possibly with the expulsion of gravel or calculi.

In *strangury* the pain is confined to the lower part of the abdomen, and is associated with urinary urgency.

In colic in the unimpregnated uterus the pain is in the hypogastrium, and it occurs in relation to the *menstrual period.*

The physiological colic of an impregnated uterus needs only to be referred to.

In *Fallopian tube* colic (one manifestation of Mittelschmerz) pain occurs in one or other ovarian region, in mid-menstrual period, there may be sudden, clear, leucorrhœal discharge, and fulness at painful side may be found on vaginal examination.

3. Colic in the alimentary canal may be more or less confined to a particular portion of its area.

In *gastric colic* pain and distension occur in the upper central part of the abdomen. The percussion note is stated to be more prolonged and of a lower pitch than over the colon. The characteristic intestinal movements are absent. Eructation of gas attended by relief, also vomiting, more freely occur.

In *intestinal colic* the symptoms previously described are characteristic, colic in the lower bowel being specially attended by tenesmus.

Appendicular colic.—In a typical case it occurs at somewhat regular intervals, say, from three weeks to three months. While at its height the pain is felt over the abdomen generally, it begins and ends in the right flank, tenderness to pressure being located in the region of the appendix. Vomiting occurs, and the patient may imagine he suffers from bilious attacks, all the more that they may recur for a period of years even. The attack lasts for a few hours, and is not in the first instance associated with feverishness. But sooner or later inflammation complicates the situation. The writer believes from clinical experience that appendicular colic is comparatively frequent, but that, as many of the attacks are mild, they are unrecognised by patient and physician alike. Being mild, the operating surgeon sees nothing of them; in severer cases he deals with them only after inflammation has complicated and obscured the clinical picture.

Etiology.—Colic, like cough, being a symptom rather than a disease, is caused by and is associated with numerous morbid conditions. It is the expression of a normal function acting in excess in response to undue stimulation, and having as its main object the overcoming of unusual difficulty or the expulsion of an irritant.

In certain individuals the intestinal musculature is more easily stimulated to excessive action than in others. There is a predisposition to colic, the same degree of stimulus having vastly different effects in different individuals. The exciting causes may be within the alimentary tube, in its wall, or outside altogether. The contents may act by their quantity or by their quality, or by both. Mere quantity by over-distension will overstimulate, *e.g.* accumulation of gas or of fæces from constipation, or from the various forms of obstruction, such as simple and malignant stricture, intussusception, volvulus, internal strangulation from bands, peritoneal adhesions and kinks, intestinal concretions, large gall-stones, coiled up bundles of worms, and hernia. Exciting quality of contents is exemplified by irritating articles of diet, themselves indigestible or containing ptomaine poison, by irritant poisons, by irritating purgative medicine, by irritating results of evil digestion. Exciting causes situated in the intestinal wall are seen in local injury, as from intussusception, hernia, etc., acting primarily by direct local effect, and, secondly, by over-distension behind; in ulceration, malignant, simple, tuberculous, or dysenteric; in inflammation, catarrhal, enteric, or peritonitic, where the pain is partly to be accounted for by excited peristalsis. Causes outside the intestinal canal find their illustration in exposure to cold, in dentition, in mental anxiety or emotion, in disease of the spinal cord as locomotor ataxia, but these are associated rather with diarrhœa or neuralgia than with colic.

It has not yet been satisfactorily demonstrated how the *poison of lead* produces colic. It probably does not act through constipation or through organic disease of the sympathetic, but whether it acts directly on the intestinal musculature or nervous ganglia, or by leading to contraction of the blood-vessels, is a moot question.

Treatment.—Whatever the cause pain demands relief, all the more that, if very severe, it may lead to serious collapse, while the excited peristalsis, of which it is a symptom, has been found to cause rupture of the bowel. When intense morphia should be injected subcutaneously, and whiffs of chloroform given until it has had time to act. When less severe a hot bath or a warm poultice applied over the abdomen, with some sedative and antispasmodic, as morphia and chloric ether internally, suffices. Pain being relieved there is time to examine quietly into the case and to determine the cause, and upon the cause depends the scientific and successful treatment of the condition. The sedative, moreover, when given in an appropriate dose, paves the way by quieting irregular spasm for that regular and harmonious action of the musculature which leads to success. In gastric colic the stomach must be emptied. If it fail to do so by its own efforts, emesis must be encouraged, or the stomach tube used.

In intestinal colic of any gravity, obscurity, or permanence, the physician should, without undue delay, associate himself with a surgeon. Where mere mechanical causes can be excluded, such as hernia, volvulus, etc., nature's attempt to empty the bowels must be assisted by the immediate use of rectal injections, and by the administration, where vomiting is not actively present, of non-irritating purgatives, such as castor-oil, calomel, or salines.

Where fæcal accumulation is within reach it is generally necessary to combine the use of the finger with the action of the enema in order to get rid of it. When legitimate efforts have failed to empty the bowel and give permanent relief, there should be no undue delay in deciding the question in consultation with a surgeon whether the abdomen is to be opened or not for the detection and removal of any obstruction, or for the making of an artificial anus above the recognised seat of obstruction.

Appendicular colic must be dealt with by removal of the appendix in a quiet interval.

Until relief is obtained, in acute cases it is practically useless to feed the patient. Ice to suck or hot water to sip in order to relieve thirst should be given.

Where recurring attacks occur from maldigestion, as in bottle-feeding of infants, prevention must be secured by proper dieting.

Where colicky attacks are caused by catarrh or by ulceration of the bowel, appropriate dietetical and medicinal treatment is necessary.

The colic said to be associated with the first stage of peritonitis, and the colics from causes outside the alimentary canal, acting reflexly, can only be relieved by sedative treatment such as morphia or the hot bath.

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A.—Inflammation and Ulceration—1. Simple Colitis.—The colon may be the seat of a simple catarrhal inflammation comparable to a gastritis or a bronchitis. If in such a case the inflammation extends low enough, a rectal examination with a speculum will show the mucous membrane to be intensely injected, of a light red colour, swollen, and secreting a thick mucus. The description of Wilks and Moxon gives a very good picture of the state of things seen after death. They mention a "case attended by discharge of mucus and blood where after death the whole internal surface of the colon presented a highly vascular soft red surface covered with tenacious mucus or adherent lymph, and here and there showing a few minute points of ulceration. The coats also were much swollen by exudation into the mucous and submucous tissues."

There are two varieties of simple colitis, viz. that which occurs either by extension from neighbouring parts or in association with some grave morbid condition, and that which is unassociated with any other serious malady. This, which has been called *acute primary colitis*, will now be described.

Symptoms.—The main symptom is diarrhœa, which may come on suddenly; there is much mucus in the stools, and often blood also, even in considerable quantities. At first there may be absolutely no fæcal matter, but as the patient improves the motions contain more fæces and less mucus. The blood is mostly fluid and but little changed, so that we may infer that it has but recently left the vessels, and has come, therefore, from the large intestine. Often the mucus is in little lumps. Triple phosphate crystals may be seen on microscopical examination, and, more rarely, oxalate of lime, cholesterin, and Charcot's crystals. The bowels may be open many times a day, and although tenesmus is not a striking feature, it may be present. A rectal examination reveals nothing abnormal except that the mucous membrane may feel a little rough. Abdominal pain is a very common symptom; it comes on in paroxysms, often associated with defæcation, but it bears no relationship to food. It is of a griping character, often very severe, and it nearly always follows the course of the colon. Between the attacks the patient may be free, but he sometimes complains of a dull pain. Abdominal tenderness is usually present; a very common seat for it is over the *sigmoid flexure*, but the whole colon, or even the whole abdomen, may be tender. There may be considerable pyrexia even when there is much diarrhœa and loss of blood. The pulse is rapid, and in a severe case small, soft, and running. The abdomen is not much distended, and sometimes nausea, vomiting, and loss of appetite are present. The tongue is furred, and the fur is nearly always a pure white, while in many diseases with which colitis might be confounded it is a brownish white. There is often much mental depression; the sufferer from this disease exaggerates trifles and takes a gloomy view of life. In some cases the neurotic element is very marked, and we are forcibly reminded of the chronic neurotic dyspeptic. Many of these neurotic patients are men who may be said almost to live for their illness, and they are usually most difficult to cure.

Simple colitis is by no means always so severe as this description might lead the reader to expect, but the difference is only one of degree. The majority of cases are mild, but, on the other hand, the diarrhœa may be uncontrollable, and the patient may die from exhaustion.

The *treatment* consists in keeping the patient absolutely in bed until the diarrhœa has stopped, the motions are well formed, blood is no longer passed, and the temperature is normal; and longer still if he has had a severe attack. Warmth to the abdomen is very desirable. He should consume nothing but milk, and should not take more than two fluid ounces at a time. The total daily amount will depend upon the acuteness of the attack and the general condition of the patient. A good way of checking the diarrhœa is to give some laudanum or chlorodyne with fifteen or twenty grains of carbonate of bismuth suspended in some mucilage every four hours; the compound kino powder is also very valuable. If these drugs fail, a starch and opium enema will often succeed. Should opium be for any reason contraindicated, compound catechu powder is very useful. But all astringent drugs should be omitted as soon as possible, for the constipation that follows colitis is often very troublesome. It is best relieved by a rectal injection of six or eight ounces of warm olive oil or a drachm of glycerine. A long holiday among new and interesting surroundings greatly aids the convalescence in neurotic cases.

The *diagnosis* is not usually difficult. The disease is known from *ulcerative colitis* by its sudden onset, by the large amount of mucus in the motions, by the fact that blood appears early, and also by the fact that under appropriate treatment the case usually yields. In England *acute dysentery* is hardly likely to lead to a mistake, but the meat-washing character of the stools, the burning pain in the rectum, the intense tenesmus, and the constant desire to go to stool even when nothing is passed, should prevent mistake. It is, however, important to remember that in England we often see severe cases of colitis in persons who have had dysentery abroad, and under strict treatment these cases recover completely. *Enterica, malignant disease* of the bowel, and *arsenical poisoning* may all give rise to errors of diagnosis.

The *prognosis* is, as a rule good, and acute cases are soon well, but in cases that have been allowed to become chronic—and often the disease has lasted many months before it is taken seriously in hand—the patients require rest in bed and milk diet for many weeks before recovery is complete; and it should be remembered that few patients are kept in bed too long, but that many are not kept in bed long enough. The more neurotic a patient is, the worse is the prognosis, and some neurotic women seem never to lose the disease in spite of most careful treatment.

That colitis which occurs either by extension from neighbouring parts or in association with some grave morbid condition need not detain us long, for it is generally marked by the diseases of which it forms a part. I have come across the following varieties:—

I have seen the colon acutely inflamed in a case in which there was *acute gastritis* due to the swelling of an acid. The stomach was in contact with the colon, and the colitis appeared to be due to direct extension of inflammation, for all coats were implicated.

Sometimes direct irritation, such as *enormous doses of purgatives*, will cause colitis, and in some cases of *arsenical or mercury poisoning* colitis is found which is probably due to excretion of the metal into the large intestine after it has been absorbed higher up in the alimentary canal.

Acute colitis may be associated with *septic* or *pyæmic conditions*. As an instance I may mention the case of a woman who had pelvic abscesses and peritonitis following gonorrhœa. She died from exhaustion. In the cæcum and for two feet beyond it the mucous membrane was sloughing, and from beyond this to the anus the colon was acutely inflamed, its walls were œdematous and thickened, its mucous surface was grayish yellow with patches of submucous hæmorrhage; the mucous membrane was separating in shreds in many places.

Acute colitis is a rare complication of *Bright's disease*, and it may be that this form should be regarded as the early stage of ulcerative colitis, which is a recognised complication of Bright's disease. This form of acute colitis is often overlooked because the diarrhœa due to it may be attributed to the purgatives so often given in Bright's disease, to uræmia, and very little stress can be laid upon blood in the motions, because patients with Bright's disease may bleed from their intestinal tract.

Colitis may in very rare cases be associated with *pneumonia*, and occasionally a *lardaceous colon* becomes acutely inflamed.

2. Membranous Colitis.—Two distinct varieties exist, one which might be termed *dyspeptic membranous colitis*, and another which is always associated with some other grave condition, or is due to direct injury, and therefore might be called *secondary membranous colitis*. It is not usual to include under membranous colitis those cases of constipation in which, when the bowels are open, shreds of coherent mucus are passed with or without hard masses of fæcal matter; such cases are common enough.

Dyspeptic membranous colitis is so called because the sufferers from it complain much of dyspepsia and they pass membranes from the anus.

The patients are usually over twenty years of age; the disease is commoner in women than in men, and in private than in hospital practice. In children it is excessively rare, for Edwards found that out of 111 cases only 6 were under the age of ten. The distinguishing feature of it is that membranes, which the patient usually calls skins, are passed from the anus. They are usually white, but may be brown from fæcal staining. In extreme cases the membrane forms a complete tubular cast of the intestine, usually varying from one to six inches long, but such tubes have been known to be much longer, and sufficiently thin and tenacious to permit of their being held up. The wall of the tube may be laminated, and fæces may be found between the laminæ, showing that they have been laid down at intervals. Fæcal matter may also be found in the tubes, the diameter of which may be anything up to 1½ inch. The thickness of the wall may be anything up to a quarter of an inch. Either with or without the tubes, shreds of all shapes and sizes may be passed, but in essential characters they are the same as the tubes. Often several pieces of membrane are passed rolled up into a ball. Under the microscope the membranes are structureless and transparent; embedded in them may be seen minute fragments of food and fæces, some cells, free nuclei, micro-organisms, phosphates, and cholesterin crystals. The cells are apparently the epithelial cells of the intestine that have undergone fatty degeneration. When the inner surface of the membrane is magnified it appears reticulate and presents at regular intervals depressions or even perforations that clearly correspond to Lieberkuhn's follicles, and sometimes it is evident that the cells lining the crypts have been cast off with them. Chemically the membrane consists of mucus, and is a coagulated secretion of the intestinal mucous membrane.

The patients who suffer from this disease are usually neurotic dyspeptics of a depressed turn of mind and liable to attacks of constipation. They are poor eaters, believing that first this and then that article of food disagrees with them, so that their diet soon becomes very restricted in choice and quantity; they are usually thin, anæmic, and complain of the cold; the tongue is pale and a little furred, the bowels are frequently rather constipated, and the constipation may alternate with attacks of diarrhœa. Sufferers from membranous colitis are as a rule taciturn; they rarely have buoyant spirits; they take a gloomy view of life, and exaggerate the importance of trifles. These symptoms and this frame of mind are constantly with the patient, but there are exacerbations from time to time during which there is much additional pain, usually griping, generally along the course of the colon, and often coming on a long while after food. During these exacerbations flatulence is troublesome, loss of appetite and constipation are very marked; the patient may complain of nausea or suffer from actual vomiting; the tongue is very furred and may be red, and the mental depression and feeling of weariness are very pronounced. After this state of things has gone on for a week, the patient notices that she passes a considerable quantity of "skins." In a mild case the patient is in the intervals between these attacks restored to feeble health without the passage of any membrane, but in other cases some but less membrane is passed in between these exacerbations of the disease. When passing membranes the patient is as a rule excessively constipated, and frequently takes enormous quantities of purgatives. The act of defæcation is often excessively painful. I have known a patient sit on the water-closet for a couple of hours suffering from agonising griping pain before a motion, consisting of enough blood and membranes to fill a half-pint measure, was passed. This patient often had to take a morphia injection during defæcation, and twice she fainted in the water-closet. As a rule, a rectal examination reveals nothing abnormal. Only in mild cases does the opening of the bowels relieve the symptoms from which the patient suffers.

Women who complain of membranous colitis are particularly prone to suffer from amenorrhœa and pelvic troubles; indeed almost every case that consults a general physician has been previously under the care of a gynæcologist. Most of these patients, if severely ill, are valetudinarians who drag out their lives passing from one health resort to another, and many of them have enteroptosis or prolapse of the intestines.

Treatment.—Between the attacks the patient should in all respects lead as healthy a life as possible. She should take plenty of exercise. Riding or playing golf is infinitely preferable to dull solitary walks taken merely for the sake of taking exercise. The diet should be ample; most of these patients decline first one article of food and then another, until at last they are not only underfed, but their digestive powers are overtaxed in one particular direction. An obviously indigestible dietary should be avoided, but otherwise the patient should partake of whatever is put before her. It should be well cooked, and the meals must be as tempting as possible; they should be at regular times. Van Noorden thinks that there should be plenty of coarse vegetables and fruit so as to mechanically act upon the colon. The patient should go to bed early, and have eight hours' sleep. All these patients are worse if they are idle. They should always have some occupation. Purgatives should be avoided, for they are particularly liable to cause indigestion; the bowels should be kept open by going to the water-closet regularly at the same time every day, by healthy living, and, if necessary, by a little abdominal massage before rising in the morning. A holiday and change of scene to some such bracing place as Switzerland or Norway is often of the greatest benefit. Indeed, these patients derive much benefit from such a holiday even when they are not passing membranes. During the period when they are passed it is particularly important not to give purgatives by the mouth; but if the constipation be extreme and the griping pain severe, considerable relief may be afforded by a rectal injection of a drachm of glycerine or half a pint of warm oil or soap and water. For a severe case rest in bed, morphia injected subcutaneously, and the application of hot fomentations to the abdomen may be necessary, but opiates should not be prescribed unless they are absolutely necessary, for not only do they increase the constipation, but sufferers from membranous colitis are just the sort of people who may have become addicted to an excessive use of these agents.

Probably future experience will show that when the patient has tried all other means of relief without success, and when suffering is so great that life is a burden, that the best treatment is to open the colon on the right side, and thus give the diseased bowel complete rest. The artificial anus may then be closed. I believe the first published case in which this treatment was adopted was one brought before the Clinical Society by Mr. Golding-Bird and myself in 1895, and since then other authors have published cases and Mr. Golding-Bird and I have brought two others before the Clinical Society. The small experience we have at present of this method of treatment appears to show that the artificial anus should be left open for at least six months. This obviously in many cases will prevent this treatment, for it is exceptional to find a colotomy plug fit so well that the patient can carry on her employment. Still the published cases are on the whole encouraging, and one of our patients appears to be permanently cured. We have found that it is quite unnecessary to wash out the bowel from the artificial to the natural anus. In more than one case the formation of membrane ceased when the bowel was attached to the colotomy wound, even before it was opened, showing apparently a reflex arrest of the morbid process. This quite accords with what we have said as to the importance of the neurotic element in membranous colitis.

Recently Einhorn has adopted the following method of treatment. During the attacks he orders rest in bed, gives some opium; between the attacks he injects eight to fifteen ounces of warm olive oil into the bowel every night. This the patient, if possible, retains. These injections are given nightly for three weeks, and are then gradually reduced in frequency till one only is given every week for five or six months.

Secondary membranous colitis is not of much clinical importance, for the maladies with which it is associated are so severe as to mask any discomfort due to the membranous colitis. As dyspeptic membranous colitis is rarely fatal, the phrase membranous colitis as used in the dead-house nearly always refers to the secondary form; on the other hand, the phrase as used in practice nearly always refers to the primary form, for patients affected with the secondary variety do not often pass much membrane.

Direct irritation may cause secondary membranous colitis, as in the cases in which it has followed *mercurial poisoning,* in which condition it is almost certain that the membranous colitis is due to the excretion of mercury into the large intestine. It may also be *septic,* as in the case of a woman who died of puerperal fever, and in whom the whole of the large intestine was buried by a greenish black membrane. Secondary membranous colitis may also be associated with *Bright's disease, pneumonia, diabetes,* and *cancer,* and it is said, too, with other specific fevers and tubercle. *Fat necrosis* of the peritoneum has been seen in cases of membranous colitis.

3. ULCERATIVE COLITIS.—The colon is frequently ulcerated as a result of typhoid fever, dysentery, tuberculosis, or malignant disease; but from time to time we meet with cases in which this part of the bowel is extensively ulcerated quite apart from any of these diseases. Observation at the bedside has shown that nearly all patients in whom this independent ulceration is found after death have during life presented such a grouping of symptoms as to enable us to predict that the colon would be found ulcerated. To this disease, with its characteristic symptoms and characteristic morbid anatomy, the name simple ulcerative colitis, or, more shortly, ulcerative colitis, is applied. It is a bad name, because the colon is ulcerated in other diseases; but it is so generally used that much confusion would be caused by any alteration of it at present.

The cause of this disease is unknown, but it is probably due to a micro-organism; and possibly different varieties of it are caused by different but closely allied micro-organisms. It appears to be more common in asylums than in ordinary practice, but otherwise nothing certainly is known of its relationships except that it is often associated with Bright's disease, but it is said in olden days to have been associated in asylums with typhus. Statistics would seem at first sight to show that it has been more commonly met with of late years than formerly, but this is probably due to the fact that we have only recently learnt to recognise it.

It is not a disease of childhood nor of old age, for the patients are usually between 25 and 55 years old; it is equally common in men and women.

Symptoms.—It is nearly always the state of the bowels which seriously directs a patient's attention to his illness. The first symptom which he remembers is usually abdominal pain, generally griping, sometimes very sharp and severe, and often sudden in its onset. Soon it disappears, only to reappear later. The duration of these painful attacks varies from a few minutes to many hours, and their alternate appearance and disappearance is very characteristic. The pain is always referred to the front of the abdomen, but its exact position varies in different cases, and also in different attacks in the same patient. Occasionally there is also pain in the back and loins; and once I have known a patient complain of such severe pain in the front and the sides of the chest that she was incorrectly thought to have pleurisy as well as ulcerative colitis. In acute cases the first attack may be excruciatingly severe, but often it does not cause much suffering; the intensity of the pain, however, increases in each succeeding attack. In the interval between the attacks the patient is usually quite free, or he may complain of a dull pain in the abdomen. It should be mentioned that the pain bears no relationship to the ingestion of food, but is commonly worse when the bowels are open. The mere presence of ulcers is insufficient to explain the pain, for it is commonly absent in typhoid fever; it is probably due to some peculiarly irritating ingredient of the contents of the intestines which stimulates the nerves exposed on the floor of the ulcer, and thus sets up irregular peristaltic contractions. In the majority of cases there is no abdominal tenderness; when this is present it is rarely intense, and most often it is especially marked over some part of the colon.

The presence of severe diarrhœa—sometimes interrupted by short periods of constipation—is almost of equal symptomatic importance to the pain; it is often the first symptom noticed, and these two symptoms are never absent throughout the whole of a case. The frequency with which the bowels are open commonly varies between two and about a dozen times in the twenty-four hours. Although the act of defæcation is often accompanied by abdominal pain, there is rarely the intense tenesmus characteristic of dysentery, nor have I heard of a patient complaining of that almost constant desire to go to stool which is so frequent in a severe dysenteric attack. If the case is mild, an occasional solid motion may be passed, but the evacuations are nearly always fluid, dark, foul smelling, and of a consistency varying between that of slime and water. So far as my experience goes, they never resemble ordinary dysenteric or choleraic stools; indeed I think they are so unlike that the distinction is probably of some diagnostic value. But it is only right to add that some of those whose work lies chiefly in asylums regard the ulcerative colitis they see as a variety of dysentery. Blood is commonly present, sometimes in considerable quantities, so that the patient describes the motions as being like red currant jelly, sometimes only in traces. It is more often fluid than solid, and is usually bright red, showing that the bleeding has been recent. Commonly it is passed with the motion, although not intimately mixed with it. The clots may be of such a shape as to suggest that they have recently come from the floor of an ulcer.

When diarrhœa is present very little fæcal matter is seen in the motions, and what there is consists of a few small lumps scattered about in the fluid. In some cases a little mucus has been observed, but it is never present in large quantities. Often shreddy masses looking very like sloughs are seen; under the microscope they are generally structureless; probably their structure has been destroyed by the action of the contents of the large intestine.

Soon other symptoms are superadded to the pain and diarrhœa. The most frequent of these, and one of the earliest, is vomiting. Often the patients say that they have been vomiting incessantly, but under careful dieting and rest in bed this usually subsides. Occasionally it is entirely absent; it bears no relationship to the intensity of the ulceration. Thirst, probably due in part to the vomiting and diarrhœa, may be very troublesome. Nausea, either with or without vomiting, may be complained of. The tongue is usually covered with a dirty white fur; but as the disease progresses it becomes red and dry with a brown fur. If the patient be seriously ill, his face has the drawn expression characteristic of abdominal disease. The abdomen is commonly distended, and peristaltic movements may be visible. A rectal examination should never be omitted, for it may be possible to feel the ulceration, and also to determine whether malignant disease is present.

The general symptoms are such as would naturally be expected. The patient gradually becomes wasted, anæmic, and excessively weak, so that he lies in bed looking bloodless, sallow, and extremely ill. The pulse is feeble, and hæmic murmurs may be heard. Irregular pyrexia is often present, the temperature ranging between 100° and 102°, but if the patient be very collapsed it may be subnormal; on the other hand, I have known it as high as 104°. Towards the end of the patient's life he sinks into the typhoid state. The cause of death is usually exhaustion, and in some cases it is distinctly accelerated by hæmorrhage. Occasionally perforation kills. It is noteworthy that, although ulcerative colitis is frequently associated with granular kidneys, sufferers from it do not often show signs of uræmia.

Prognosis.—This is very grave—so grave, indeed, that when patients appear to recover from it, it is quite an open question whether the diagnosis was correct; still sometimes patients with every symptom of ulcerative colitis do get well. The patient is usually dead in eight weeks from the commencement of the illness. The symptoms which especially indicate a speedy termination are great tympanites, much loss of blood, very profuse diarrhœa, and occasionally a high temperature. Some observers state that it is the cause of death of a third of the patients in asylums.

Diagnosis.—I think there is little doubt but that ulcerative colitis is a distinct disease from dysentery, but the matter cannot be absolutely settled while our knowledge of the bacteriology of the two diseases is so imperfect. Ulcerative colitis does not attack all ages; the bowels are never open 50 or 100 times a day; there is no severe tenesmus; the motions are not dysenteric, vomiting is common, and Bright's disease is often present. In England the two diseases are not likely to be confounded, for acute dysentery is not common, and chronic dysentery is too long lasting to be confounded with ulcerative colitis. In actual practice the difficulty of diagnosis is usually to separate ulcerative colitis from *malignant disease of the large intestine*, from *intestinal obstruction*, and from some form of *primary anæmia*, but a mistake is usually due rather to a faulty examination of the patient than to any real difficulty.

Morbid Anatomy.—Any part of the large intestine may be affected, and generally the ulceration has no special distribution. In an extreme case the muscular coat is exposed, the floor of the ulcers can be seen to distinctly consist of bundles of muscular fibres, and the ulceration is so extensive that only islets of mucous membrane are left here and there; often they are considerably swollen, and consequently they look taller than they otherwise would; and frequently they are more or less stalked because of the ulceration which undermines them. The result of this is that a careless observer takes the islets of mucous membrane for polypoid growths, and the exposed muscular coat for the natural level of the colon. The vessels of the mucous membrane are dilated. The number of ulcers varies from one or two to several dozens; but while ulcers of recent date are often more or less circular, and vary in size from a pea to a five-shilling piece, by the time death occurs their shape is usually very irregular, and they have run one into another. Attempts at repair are very rare. Perforations may be very numerous. In some cases the small intestine is affected as well as the colon, but this is rare, and the process is much more severe and apparently of longer standing in the colon. The liver is often fatty.

Associated Diseases.—I have already mentioned that the disease is common in asylums, and there patients dying from ulcerative colitis are frequently found to have pneumonia. Gemmel thinks the onset of pneumonia always fatal. Outside asylums, chronic Bright's disease is the condition most frequently associated with ulcerative colitis, and even in Gemmel's asylum, out of eighty fatal cases of ulcerative colitis the kidneys were cirrhotic in thirty-five. One of Bright's original cases of albuminous urine is that of a woman aged 40, in whom the kidneys were hard, rough, and lobulated, and the large intestine was ulcerated throughout. Pyæmic hepatic abscesses are very rare, but occasionally a single large hepatic abscess occurs in association with ulcerative colitis.

Treatment.—Unfortunately this avails but little. The patient must remain in bed and be kept warm. If the pain be very severe, hot abdominal fomentations are comforting. The diet should consist of milk, and often a little brandy is required. Bearing in mind the abdominal pain, and that the diarrhœa is often excessive, opium is probably the best drug to prescribe. In a severe case the patient should be kept well under its influence; it matters little what preparation is given, care of course being exercised if the patient has chronic Bright's disease. Half or three-quarters of a pint of a saturated solution of boracic acid at about 100° F. may twice a day be let to run slowly into the bowel through a long rectal tube introduced as far as possible while the hips are raised. Gemmel uses salol dissolved in turpentine in the same way, and he attaches much importance to the administration of quinine by the mouth. The administration of Ol. eucalyptus, in capsule form (m. v. t.i.d.), has been found of service, and deserves a further trial. Perhaps in suitable cases it would be a good thing to open the colon on the right side, to let the fæces be discharged through the artificial anus, and then to syringe through from it with some antiseptic solution.

OTHER VARIETIES.—*Follicular ulceration* is of very little clinical importance, so it need not detain us long. It begins by an accumulation of small round cells in the solitary follicles, which therefore swell; later they rupture and the ulcers are formed. These ulcers do not extend deeply; they have sharply cut edges, and in a well-marked example the gut is quite honeycombed with them, varying in size from a hemp-seed to a large pea. I have never met with any case in which there was an attempt at repair, nor on the other hand have I ever heard of perforation, for the floor of the ulcer is always formed by the muscular coat.

The patients in whom follicular ulceration is found have always died of some other disease, often of the gastro-intestinal tract. Thus I find that of ten adults, one had dysentery, one cancer of the rectum, one membranous colitis, one typhoid fever, and one femoral hernia. Follicular ulceration of the colon occurs about once in every 500 post-mortems in hospital practice. It is never diagnosed during life, for the symptoms of the fatal disease quite overshadow any that might be attributable to the ulceration. It is proportionately more frequent in children than in adults. Thus Holt met with follicular ulcers in the intestine in twenty out of seventy fatal cases of infantile non-tuberculous diarrhœa. They were never seen in cases that had lasted less than a week, and the proportion of follicular ulceration was highest in those that had lasted more than ten weeks. When in the small intestine they were always most numerous near the cæcum.

Vascular ulceration of the colon is chiefly of pathological interest. There are two varieties of it—venous and arterial. The venous form is by no means uncommon, and is probably due to the same cause as venous ulceration elsewhere. The arterial is due either to blocking of one of the mesenteric arteries by an embolus, to arterial thrombosis, or to atheroma, especially that of the aorta just at the origin of the mesenteric arteries. None of these are sufficiently common to call for notice here.

Hæmorrhagic Ulceration.—In some diseases submucous extravasations of blood take place into the colon, and these no doubt may break down and lead to ulceration. Hæmorrhagic ulcers are usually small, shallow, rounded, and discrete, although occasionally two or three may run together; submucous hæmorrhages may commonly be seen in their neighbourhood. Hæmorrhagic ulcers are by no means uncommon in those who have had Bright's disease. This is what might be expected, for we know that persons with Bright's disease are particularly liable to hæmorrhage from any part of the body. They may occur too in other diseases, such as purpura, scurvy, and other forms of anæmia.

Trophic Ulceration.—It is by no means proved that the intestine is ever ulcerated as a result of lesions of the central nervous system, but several cases have been put upon record—and quite recently I have seen one not yet recorded—in which intestinal ulceration was associated with disease of the spinal cord. The following is an instance: A man had complete paraplegia as a result of a fracture of the spine in the lumbar region. There were numerous small round ulcers occupying the whole of the colon. Many more cases will have to be observed before the question of trophic intestinal ulceration can be settled, but as a lesion of the spinal cord can lead to an ulcer of the lower extremities, it is not unlikely that it might lead to an ulceration of the intestine.

DILATATION OF THE COLON.—Cases in which the large intestine is dilated may be divided into four groups.

The *first* contains those in which the distension is entirely gaseous, is not due to any obstruction, and is only one symptom of some other illness. It is often seen in association with peritonitis and typhoid fever, and may occur in almost any severe illness. When extreme it is of great importance, for it adds considerably to the danger of the original disease, and greatly hampers the movement of the heart and lungs. The abdomen is very distended and tense, it hardly moves on respiration, it is hyper-resonant on percussion, and the tympanitic note extends well into the flanks, the liver is pushed up and the splenic dulness is obliterated, the patients are nearly always constipated and do not pass much flatus per rectum, but they complain of borborygmi which may be audible to bystanders. The gas consists chiefly of carbonic acid and hydrogen. The only difficulty of diagnosis likely to occur is in the distinction of this condition from those rare cases in which there is gas in the peritoneal cavity.

The treatment of tympanites is very difficult and uncertain. Various carminatives, especially the aromatic oils, are often recommended, but although I have often given them, I have never seen them to be of any use in serious gaseous distension of the large intestine. Enemas give a better chance of success; those of asafœtida and turpentine are the most useful, but we naturally shrink from the use of large enemeta in typhoid fever or peritonitis. Puncture of the bowel has been recommended. It should be done with a sterilised trocar and canula, but it often fails to give relief, for the gas soon collects again. Acupuncture should never be employed, for it often leads to an escape of gas into the peritoneal cavity.

The *second group* contains those cases in which dilatation of the colon is due to some solid substance within it. Concretions consisting of vegetable fibres, hair, and other foreign bodies are sometimes found in the insane. A gall-stone may rarely block the colon, but in the immense majority of cases in which it causes intestinal obstruction it stops in the lower part of the ileum. Distension of the large intestine by fæcal matter is of great importance, and fæcal impaction is a common cause for intestinal obstruction, under which heading it will be found described.

The *third group* contains those cases in which the dilatation of the colon is due to some organic obstruction in front of the dilated duct. The muscular coat of the dilated gut usually undergoes hypertrophy. The intestinal contents retained behind the obstruction set up some colitis, which may proceed to ulceration, and so the well-known distension ulcers are produced. These may be very numerous. For example, one case is described thus. "The whole of the colon above the stricture was distended and worm-eaten by small ulcers." Sometimes this leads to perforation and consequent peritonitis. It is curious that occasionally the ulcers may be a long way behind the obstruction. This colitis may be difficult to treat after the obstruction has been overcome. The dilation behind an obstruction may be very great and lead to much abdominal distension. By careful watching of the peristaltic movements, by percussion, and by palpation we can often make out whether it is large or small intestine that is dilated, but it must not be forgotten that a distended large intestine may be bent down towards the pubes like small intestine, or a distended small intestine may be as large as a normal large intestine.

The *fourth group* contains those very rare cases of so-called idiopathic dilatation of the colon. A few cases which have been recorded as examples of this condition are really instances of fæcal impaction leading to great dilatation of the colon behind the impacted mass. Putting aside these cases, a well-defined group exists among those of *idiopathic dilatation of the colon*, and the characteristic of this group is that the dilatation is of the sigmoid flexure. It may be enormous, filling nearly the whole abdomen, displacing the liver upwards, and resembling either an enormously dilated stomach, or so bent on itself as to form two huge sacs lying vertically side by side in the abdomen. It may measure two feet in circumference. When the dilatation is not of the sigmoid flexure it is usually near it. It has been suggested that the dilatation in all these cases is due to kinking, set up by the presence of impacted fæces, but this suggestion is probably not correct, for many of these patients do not give a history of constipation; fæcal impaction is very common, but the condition under discussion is excessively rare, and, lastly, often at the autopsy the distended bowel was not full of fæces. Probably *idiopathic dilatation of the colon* is allied to those cases of dilated stomach in which there is no obstruction at the pylorus, and this is especially likely to be true of those cases in which the dilatation is confined to the sigmoid flexure.

The symptoms of idiopathic dilatation of the colon can be very briefly described; the condition is much commoner in males than in females, and sometimes occurs in young children, having in them probably existed since birth. The distension of the intestine is very great, and percussion shows that it is chiefly due to gas. Shortness of breath is a cause of complaint, and the patient may be livid because the diaphragm is pushed up and the breathing hampered. The upward displacement of the heart gives rise to palpitation. The splenic dulness is obliterated, the hepatic dulness is diminished, and the liver cannot be felt. The abdomen is always distended, sometimes enormously so, and then it may be tympanitic all over; usually neither coils nor peristaltic movements are visible. In very rare cases the pressure has been so great as to lead to œdema of the legs, scrotum, and penis, and to albuminuria. Most of the patients for some time, often for years, have had some difficulty with the bowels, constipation supervening from time to time; but this is not usually very serious and has yielded to purgatives or enemas. After death, both layers of the muscular coat are found to be hypertrophied, and there is often some ulceration of the dilated mucous membrane. The distended bowel contains some semi-solid fæces and an enormous quantity of gas. Of the recorded cases all the patients except one have died: occasionally death is due to perforation of an ulcer, but more frequently the cause of it is by no means clear, and then it may be sudden.

The treatment has hitherto been very unsatisfactory. Probably the best course would be to open the bowel above the dilatation, wash out the dilated part, and let the fæces pass for some time through the artificial anus, and so give the affected part rest.

SACCULATION OF THE COLON is not a common condition. The diverticula are usually the size of a pea, but they may be large enough to receive the little finger, and half an inch in depth. They are most frequent in the descending colon, sigmoid flexure, or upper part of the rectum. Usually they are very numerous, are placed close together, and contain a little fæcal matter. They are to be regarded as hernial protrusions of the mucous membrane through the muscular coat. They are found for the most part in elderly people. Only once have I heard of their giving rise to symptoms, and that was in a case in which the perforation of one of these sacculi lead to the formation of an abscess between the left kidney, spleen, and ascending colon.

INJURIES OF THE COLON.—*Laceration of the Colon.*—Sometimes, but very rarely, this is due to an enema tube or bougie, but it is not very infrequent for a nurse or a doctor to be excessively afraid that the administration of an enema has led to perforation of the bowel; for in elderly people the evacuation of the bowel by an enema is often accompanied by much faintness and even by syncope. The pulse may become quite small and the patient appear collapsed. Elderly or feeble people should never be allowed to get out of bed for the evacuation of the bowels after the administration of an enema unless they are in the habit of using enemata.

The kinds of accident that most frequently damage the intestine are: being run over, kicked in the abdomen, or stabbed there. The small intestine is much more often affected than the large. (See "Abdomen, Injuries of.")

Perforation of the Colon from without.—All sorts of abscesses may burst into the colon, thus iliac and psoas abscesses, localised peritoneal collections of pus, hepatic abscesses, appendicular abscesses, splenic abscesses, ovarian abscesses, suppurating growths, abscesses of the gall-bladder, and hydatids of the liver may all burst into the colon. This vent is usually recognised by the fact that the original tumour becomes smaller, and there is a discharge of pus by the bowel.

MALFORMATIONS OF THE COLON.—These are excessively rare, but they depend for the most part upon malposition of the cæcum. It will be remembered that the first position of this structure is outside the abdomen, then inside just under the umbilicus, then to the lower part of the left side of the abdomen, next near the cardiac end of the stomach, later under the liver, and lastly in the right iliac fossa. The cæcum may remain in any of these positions, while the colon continues to grow. This naturally gives rise to various malformations.

METALLIC PIGMENTATION OF THE COLON.—Sometimes lead may be deposited in the tissues of the colon. Thus a man who had lead colic died from chronic Bright's disease. The whole of the cæcum and large intestine was very black. The colon contained ·0086 per cent of lead. In this case as in others the pigmentation at the cæcum began very abruptly. There is nothing to show that lead is not deposited in the colon by the same process as leads to the formation of a blue line on the gums.

In very rare instances mercury and bismuth may cause black pigmentation of the colon.

MALIGNANT DISEASE OF THE COLON.—A primary growth in the colon is almost always a cylindrical celled epithelioma; it is usually circumscribed to a small spot, and then by its contraction sets up intestinal obstruction. Occasionally, however, the growth is soft and medullary, then it breaks down in the centre, and intestinal obstruction does not arise. Thus there are clinically two groups of cases of malignant disease of the colon—those without and those with symptoms of intestinal obstruction.

A post-mortem examination on the group of cases in which there is no intestinal obstruction reveals that several inches of colon are affected with growth, usually forming an obvious tumour visible directly the abdomen is opened. On cutting into this mass it is found that the normal channel of the intestine is replaced by an elongated cavity with black, ragged, sloughy walls, but passing into healthy colon above and below. I have known the whole of the transverse colon to be converted into such a mass. The contents of the cavity consist of a foul mixture of fæces, blood, and necrotic growth. If the sigmoid or cæcum is affected this mass may grow into the subjacent bone; if it is in other parts of the colon, it may extend into the stomach, liver, or kidneys. This state of things during life may give rise to no symptoms; on the other hand it may be easy during life to detect a tumour, especially if it is in the transverse colon or the cæcum. The patient may pass large quantities of foul, dark gray, or black fluid motions, in which, perhaps, fragments of growth and small blood-clots are seen. Sometimes between the passage of such motions the bowels may be constipated. The patient suffers pain, he wastes, and may even die before any secondary deposits show themselves. When the *tumour is in the cæcum or the sigmoid*, it may cause œdema of one leg and thrombosis of the external iliac vein, and when it is in the *transverse colon*, we must carefully distinguish it from an enlarged liver, carcinoma of the greater curvature of the stomach, or an omentum puckered up by some form of chronic peritonitis. The implication of other organs by direct spread of the growth rarely gives rise to any symptoms by which it can be recognised. In very rare cases of carcinoma of the large intestine a fistulous tract is formed, which opens either into the stomach, the duodenum, or some adjacent coil of small intestine. In rare instances the carcinomatous growth ulcerates directly into the peritoneal cavity.

We now pass on to the description of the second group of cases of malignant disease of the colon, namely those in which the *growth produces obstruction*. This variety of growth is usually limited to a quite small portion of the bowel, and forms an annular band, encircling the whole circumference of the affected part of intestine. The gut from the outside appears suddenly constricted, as though some one had drawn a tight string around it. The stricture is therefore quite narrow, and the peritoneal covering of the gut is usually thickened just over the stricture. From the inside the stricture appears as an annular and contracted deposit, the surface of which is irregularly ulcerated. The stricture may be extremely narrow, so that often the wonder is that the patient has presented so few symptoms. The effects that occur behind the stricture of the bowel have already been described; they are dilatation with hypertrophy of the colon and distension ulcers. The presence of these makes life hazardous. I remember a case in which a surgeon was to have operated on a patient one evening, the operation was put off until the next morning, but in the night the patient died from perforation of a distension ulcer. On the other hand, in very rare cases the opening of a distension ulcer into some adjacent viscus, *e.g.* the bladder, has relieved the symptoms of obstruction, or an abscess may form around the distension ulcer; this abscess bursts externally, and thus a fæcal fistula is formed.

With regard to the locality of stricture of the colon, Treves gives the following table of 100 cases:—

Sigmoid flexure	58
Descending colon	11
Splenic flexure	7
Transverse colon	7
Hepatic flexure	9
Ascending colon	2
Cæcum	6
	100

The *symptoms* of malignant stricture of the large intestine are as follow: the patient, who is usually a man of over fifty, says that for some time past he has been losing flesh, and that he has been liable to attacks of abdominal pain, not always severe, and, to begin with, only coming on at long intervals. The attacks get more and more frequent, and gradually there may be constant pain with exacerbations. Concurrent with these symptoms difficulty of keeping the bowels regular appears. There are attacks of constipation which are at first easily relieved by aperients, but as time goes on the patient has to take more and more aperients, until at last he comes to a doctor because nothing that he takes will unlock the bowels, and then he begins to suffer from abdominal distension, nausea, with sometimes actual vomiting, loss of appetite, and furred tongue. But this by no means completes the picture, for one of the most striking things about malignant disease of the large bowel is that these attacks of constipation often alternate with attacks of diarrhœa. This is due to the fact that the retention of fæces above the stricture sets up a colitis. Thus it follows that irregularity of the bowels is one of the striking features of this disease. The motions are often very characteristic. Thus when solid they may be altered in shape and also of small circumference, owing to having passed through the stricture; occasionally the patient describes them as tape-like or pipe-like. Too much stress is, however, usually laid upon the shape of the motions, for it must be remembered that their shape is for the most part acquired in the rectum. But when diarrhœa is present, the motions are commonly foul-smelling, contain mucus and, it may be, necrotic fragments of growth. Whether there is constipation or whether diarrhœa is present, blood is often seen in the motions. The rectum is often stated to be ballooned, that is to say, when the finger is introduced through the anus, the rectum is found to be empty, and so dilated that the finger can scarcely reach the walls, which are perfectly smooth. The cause of this condition is quite unknown; it is not always present in malignant disease of the colon, and it may be met with in other conditions. The abdomen is often very distended, and visible coils of intestine may be seen contracting through the abdominal parietes. These are often more easily visible the longer the case, for hypertrophy of the muscular coat of the distended bowel renders them especially conspicuous; but it is by no means necessary that the bowels should be hypertrophied for them to be visible, for I have seen them well marked when obstruction had only existed twenty-four hours. Sometimes a tumour can be felt through the abdominal parietes. This is more often fæcal accumulation than growth. If the growth extends low down enough it may be possible to feel it per rectum, and it may be that attention is directed to the fact that the bowel cannot hold a copious enema. Frequent rumbling and gurgling signs are heard in the abdomen. In the group of cases now being described, intestinal obstruction occurs sooner or later, and it is important to remember that even in a chronic condition, such as malignant disease of the colon, the signs of intestinal obstruction may come on acutely, and as, further, the signs of malignant disease of the colon may be unobtrusive until acute obstruction sets in, serious mistakes in diagnosis are by no means uncommonly made. Thus I have seen a case which was really one of acute intestinal obstruction due to malignant disease of the colon called acute peritonitis; but as a rule the intestinal obstruction is chronic, and the patients give a clear history of malignant disease of the colon. Sarcomata and innocent tumours of the colon are too rare to call for mention here.

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Colotomy.

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THE operation of colotomy, or the formation of an artificial anus, is undertaken for certain grave maladies of the rectum and of other parts of the large intestine, and the modes of executing the operation, and the place at which it is to be performed, depend themselves on the nature and the degree of gravity of the ailment. Formerly colotomy was regarded as an operation to be postponed until obstruction was complete; now it is more generally used; it should not, however, be employed without due consideration.

INDICATIONS FOR. Cancer of the rectum and of other portions of the large intestine, and *strictures* and *ulceration*, whether tuberculous, syphilitic, or dysenteric, etc., sometimes combined with *extensive fistulæ*, are the main diseases that call for colotomy, provided that intestinal anastomosis or resection of the stricture is found to be impossible. The chief conditions, besides obstruction, which may necessitate colotomy, are great *pain*, severe *hæmorrhage*, and intense and persistent *diarrhœa.*

When the growth or stricture causing the obstruction is within the rectum its exact position can be felt, and the surgeon can determine whether to adopt other means or to resort to colotomy; but when the obstruction is in any other portion of the large intestine, unless a growth can be definitely discovered, it is wise to make an abdominal exploration, so as to find out the position of the obstruction and the best mode of dealing with it.

In some forms of cancer of the rectum and other forms of ulceration the great pain caused by fæces passing over the growth or into fistulæ about the buttocks of itself necessitates colotomy.

The severe and dangerous hæmorrhage caused by the passage of fæces over very vascular growths may sometimes demand colotomy.

Colotomy is frequently required for persistent diarrhœa arising from cancer of the lower part of the sigmoid flexure and of the upper part of the rectum, or from syphilitic or tuberculous ulceration, not only of the lower, but also of the upper parts of the large intestine. In such ulcerations, tuberculous, syphilitic, or dysenteric, colotomy, performed above the diseased portion of the gut, will stay the diarrhœa.

Colotomy falls under three heads, namely *inguinal colotomy*, left or right, *lumbar colotomy*, left or right, and *transverse colotomy.* The *left inguinal* and the left lumbar operations are far the most frequent, and a general preference may be given to the former. The opening being in front in inguinal colotomy, it can be attended to by the patient much more easily than is the case in the lumbar region, and a pad or truss can be more readily adjusted. The importance and possibility of being able to procure a good spur, and so preventing fæces from passing below the colotomy opening, are strong points in favour of the inguinal method. Especially is this the case if the colotomy has been done for the relief of pain, hæmorrhage, or diarrhœa.

Again, a spur can always be made in the inguinal region, but this is not so in the lumbar region, as often the gut is too firmly fixed to the loin, and at too great a depth from the surface wound.

For transverse, right lumbar, and right inguinal colotomy the rule is to perform the colotomy immediately above the stricture, or stricture with ulceration, if such be clearly indicated. In uncertain cases a median abdominal exploration should first be made, and when the seat of the obstruction is found, the colotomy should be done above that; thus for disease about the splenic flexure of the colon, transverse colotomy is the best; for disease at, or extending up to the hepatic flexure, right lumbar colotomy is preferable; for disease lower down, right inguinal colotomy should be chosen. If the exploratory operation has not had definite results, right inguinal colotomy is the best, so that the surgeon may work well above the diseased part of the large intestine.

OPERATIONS

(*a*) *Left Inguinal Colotomy.*—In left inguinal colotomy, about an inch and a half inside the left anterior superior spine of the ilium, and parallel with Poupart's ligament, the skin and cellular tissue are divided by an incision not more than two inches in length, and often less. The muscles are divided down to the subserous areolar tissue, which is torn through, the peritoneum is opened, the finger is introduced into the opening, and the deep structures are divided with scissors up to the extent of the skin wound.

My former practice was to stitch the parietal peritoneum to the skin; now I think it wiser not to do this, as it was shown by the late Dr. Greig Smith that a much better union is obtained when the peritoneum covering the gut unites to muscles and cellular tissue, than when peritoneum becomes united to peritoneum.

The gut is sought for and brought to the surface, and is at once recognised to be large intestine by its longitudinal bands and appendices epiploïcæ. While it is held out of the wound the mesentery fixing it to the back of the abdomen is sought for. Then through the mesentery, just as it joins the gut, a pair of Spencer Wells's forceps (closed) are passed, and these forceps, perforating the mesentery, are allowed to rest across the wound in the abdominal wall. They thus support the gut, and prevent it from slipping back again into the abdomen. The handle of the forceps should point toward the middle line; the apex or point of the forceps should point outwards.

A suture is then passed through the serous and muscular coats of the bowel, and through the skin at the upper and lower angle of the skin wound. If necessary, three or four other sutures may be used in like manner to unite the gut to the skin about other parts of the wound.

I now use forceps rather than a glass rod or the mesenteric stitch, as they are not so likely to slip out, as may happen when the glass rod is used, or tear through prematurely, as is at times the case with the mesenteric stitch.

It is most important that these forceps should be kept through the mesentery behind the gut for at least ten days, after which they are removed.

In from twenty-four hours to three days after the operation, the gut is opened by a transverse incision over the forceps and parallel to them. It should be made only long enough so as to allow flatus or liquid fæces to pass out.

In about ten days' time, or longer, if the union between the gut and the wound does not appear to be very strong, the entire gut is cut through in a transverse direction, the incision going through the opening made to let out flatus and fæces. This cut is carried down to the clip forceps through the mesentery—in fact, it absolutely divides the gut across, and thus frees the forceps. By cutting down on to the forceps, one can make sure that the gut is really divided in two, for, as previously shown, the forceps at the original operation are passed through the mesentery behind the gut.

By the cutting the gut transversely there is no pain, and little or no bleeding, as the nerves and vessels run transversely round the gut.

The essential point of the operation is to prevent fæces passing below the artificial inguinal opening, and to effect this the gut is fixed up by the forceps through the mesentery—in other words, a spur is procured. Unless this is obtained there will not be an artificial anus, but a fæcal fistula, beyond which fæces pass into the distal portion of the gut. Thus the fæcal irritation is continued, and the operation has failed in one of its objects. But when a proper spur is made, fæces pass through the inguinal opening only, and cannot enter the distal end of the intestine.

(*b*) *Right inguinal colotomy* is performed by the same incision as on the left side, but this incision should be lower down and nearer to Poupart's ligament. The cæcum is the region to be opened. The question of a spur can never arise.

In cases demanding this operation, when the cæcum is very distended, Paul's tube is of very great service. The peritoneal cavity is opened, and the cæcum is drawn into the wound and incised; a small Paul's tube is at once inserted, and the cæcum tied around it. To the free end of the glass tube a drainage tube is attached to carry the fæces away to some vessel remote from the wound. More stitches are used to fix the cæcum to the edge of the wound. The Paul's tube is removed in a few days, when the cæcum is well glued up to the surface. Paul's tube should be used in all parts of the large intestine when distension is great and it is necessary to open the gut at once.

(*c*) *Lumbar colotomy* can be performed in several ways, the direction of the incision and the mode of fixing up the gut being the main subjects of difference.

The best incision for finding the colon is one with its centre half an inch posterior and midway between the anterior superior and posterior superior spines of the ilium, and midway between the last rib and the crest of the ilium. From this point Callisen used a vertical incision, but the length of this is limited, and it is not easy to work down upon the gut. More room is given in difficult cases by Amussat's transverse, and Bryant's oblique incision.

Half an inch behind the point described, and with its centre over the chosen spot, an incision (either transverse or oblique) is made, not longer than two inches, for otherwise the gut may be missed. The skin and cellular tissue are divided, and the muscles exposed and quickly divided till the fascia lumborum is reached. This is opened, and the quadratus lumborum is exposed at its anterior edge. The edges of the wound are retracted, and the fat around the kidney and the fascia lumborum is opened up. Then the gut will bulge into the wound, if it is distended and has no mesentery. In straightforward cases it will be recognised to be the colon by its being uncovered by the peritoneum; for if the peritoneum is opened, peritoneum will be seen surrounding the gut, and the longitudinal bands, and the appendices with these, will also be visible. The colon is then brought to the surface, and stitched all round to the skin wound with interrupted sutures, which should pierce the muscular coat only, and not perforate the gut.

But rarely is this actual presence of the colon made absolutely certain without opening the peritoneum, and the difficulties arise from the position of the intestine in relation to its peritoneal covering and length of mesentery. In what was formerly, but erroneously, supposed to be the general position, the peritoneum covers only half or two-thirds of the circumference of the gut, leaving the posterior part uncovered, with the intestine bound down to the loin. If no longitudinal band is seen, there is a danger of opening the small intestine, or even the stomach, imagining that the large intestine is being dealt with. I am therefore strongly of opinion that one should intentionally make a small incision in the peritoneum towards the inner part of the wound, and, by finding intestine with longitudinal bands or appendices epiploïcæ, become certain that the large intestine has been reached.

In a second condition, where the colon is entirely surrounded by peritoneum and has a mesentery, it cannot be reached, nor can the longitudinal bands be seen, without first opening the peritoneal cavity. By the index finger inserted into the peritoneal cavity the gut can be felt and hooked up and sutured, as above described, into the loin wound.

In a third condition the mesentery is very long, and the intestine may alter its position in the abdomen so as to be on the side opposite to that in which the incision is made. Prior investigations failing, the external wound, and the peritoneum to a corresponding extent, must be enlarged so as to admit the hand, and a search is made for the splenic or for the hepatic flexure of the colon. From one of these points the colon can be found, and brought to and fixed to the skin wound.

Right lumbar colotomy is performed in the same way and with the same difficulties, as the mesentery is usually fairly long, and therefore it is most important to open the parietal peritoneum to make sure that the large intestine is being dealt with.

(*d*) In the infrequent operation of *transverse colotomy* the rectus muscle just above the umbilicus is exposed and the posterior part of its sheath incised, and the peritoneum is picked up and divided, and then, as in inguinal colotomy, the large intestine must be found and recognised by its longitudinal bands, and then pulled forward and fixed outside the abdomen. The gut is secured to the skin by sutures through the peritoneal and muscular coats. The incision should always be large enough to admit the introduction of the hand into the abdomen, to make sure that the colotomy is being done above the ulceration or stricture. Then the lower part of the wound is brought together as in abdominal section, the upper two inches are treated as in inguinal colotomy, and through them the transverse colon is brought and fixed. When a spur is required to prevent fæces passing beyond the opening, a clip can be put behind the gut as in the inguinal operation.

AFTER-TREATMENT.—For all the forms of colotomy the after-treatment, *mutatis mutandis*, is the same. After the operation the patient is put back to bed, lying on the back, with the head low and a pillow placed between the knees. The use of opiates is dissuaded, unless necessitated by very great pain, restlessness, or severe purging. For the first twenty-four hours as little food as possible is given, the preference being for nutrient enemata, when possible, or soda and milk, weak tea for washing out the mouth, and brandy and water in rare cases of faintness.

Several points should be noticed on the first visit on the evening after the operation. Pain in the back may be remedied by turning the patient on the side opposite to that on which the colotomy has been performed, not on the same side. Any probable bronchitis may be prevented by the patient being propped up in bed. If wind causes much distension or tympanitis, the bandages should be loosened; or, if necessary, the dressings may be removed and the gut slightly punctured with a lancet, so that flatus may escape. The question of the passage of urine should be seen to.

The next day the dressings are left undisturbed, and the diet is still kept meagre and liquid, beef-tea being permissible.

The second day after the operation the dressings are removed, and the gut is opened by scissors for about one or one and a half inches in a transverse direction of the gut to allow of the escape of wind or fæces. This may be done earlier or deferred, according to circumstances. Any small vessels that bleed are clipped or ligatured; slight oozing of blood is allowed to clot, absorbent wool is applied to the wound, and dry gauze is used as a covering. After this, diet can be freer, fish being allowed first.

The day after the gut has been opened the bowels should be made to act, castor oil or liquorice powder being administered. Any impaction or hard fæces that block the way should be carefully broken up with the finger and olive oil injected. The wound is dressed with wool soaked in any antiseptic. Thenceforward the bowels must be carefully kept open.

In ten days' time, when the bowels have been well emptied, the gut should be cut across as shown in the description of the inguinal operation.

A little later the patient may rise from bed and lie on a sofa, with the colotomy opening dressed with ointment and supported by a pad of wool. Any tendency to contraction about the proximal opening of inguinal colotomy is corrected by the daily passage of the finger into the upper opening, the lower one being allowed to contract as much as possible. Old fæces lodged in the lower bowel may cause rectal irritation. These should be disposed of by irrigating with water into the lower of the two orifices towards the rectum, and then from the rectum through the lower orifice. In a short time the lower portion of the gut will contract and become merely a passive tube.

In about three weeks the patient can walk, wearing either a truss or abdominal bandage. The bowels should be made to act in the morning, a kidney-shaped bowl being placed under the colotomy opening. After left lumbar or left inguinal colotomy the motions are usually quite solid. After transverse, right lumbar, or right inguinal colotomy, through which the action of most of the large intestine is stopped, the colotomy opening being so close to the small intestine, the fæces are consequently liquid, or semi-liquid. This after-result tells against the three higher operations, and points to the advisability of doing the colotomy as low down the large intestine as is consonant with the position of the disease.

Colour Vision.

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Physiology of Normal Colour Vision.—The complex subject of "colour vision" lies on the borderland between the realms of Physics and Physiology, and an intelligent appreciation of its many problems demands on acquaintance with both the physical and physiological sides of the question.

Physical Considerations.—The various physical forces in nature which emanate directly or indirectly from the sun are regarded by modern physicists as the expression or outcome of undulations or wave-like motions propagated in the hypothetical all-pervading medium—the ether. These undulations vary considerably in length and frequency, and in the associated physical effects which they produce upon animate and inanimate nature.

Ascending the scale of ethereal disturbance from the long waves of comparatively slow oscillation to the shortest and most rapid undulations, modern physicists assume the existence (in sequence) of ethereal rays possessing electro-magnetic, thermic, photogenic, and actinic or chemical properties. The first and last groups of this series appear (in the present state of our knowledge) to be devoid of effect on the human organism, or, at least, we do not possess any specialised receptive sense-organ conferring on us a conscious knowledge of their existence. The highly specialised nerve-endings of the skin and retina, however, provide the appropriate receptive channels for those physical stimuli which, by some peculiar and unknown transformation therein, endow us with the faculty for the conscious perception of heat and light. At present we are only concerned with the photogenic ethereal rays, in so far as a consideration of them is essential to the comprehension of the physiology of normal colour vision.

As above premised, the sun is the great source of natural light in our universe, and the whole of the photogenic rays thence emitted are collectively appreciated by us as the solar white light. This apparently simple light is, however, of a composite character, for by means of the spectroscope it can be analysed into several component and further indivisible parts, constituting the defined colours of the spectrum with the multitudinous variety of hue or colour-tone resulting from their gradual transition into each other. In the light of the now universally accepted undulatory theory, this phenomenon of spectral colour-dispersion depends upon the constitution of the solar white light, which is composed of many groups of ethereal rays corresponding to the individual colours, and varying in their vibratory rate and wave-length, and accordingly in their refrangibility by refractive media. Colour, therefore, is the physical equivalent of wave-length and frequency, and in accordance with these attributes, the related rays produce correspondingly different impressions upon the receptive retina, which the interpretative mind appreciates as variation in colour.

It has been experimentally determined that the range of our appreciation for photogenic waves begins when the ethereal vibrations number about four hundred billions per second, and the wave-length is about 750 $\mu\mu$[1] (which is equivalent to the sensation of red), and continues through the series of increasing vibrations (represented by orange, yellow, green, blue, and indigo) until the rate of oscillation has increased to about eight hundred billions per second, and the wave-length has inversely diminished to about 380 $\mu\mu$, when the sensation of violet is produced. Beyond this point their perception as light ceases, and they manifest their existence by the possession of actinic or chemical properties.

Upon this compound character of the solar light the varied phenomena of colour depend. Colour is not (as was formerly supposed) an inherent quality of the objects around us, but is a purely subjective sensation produced in the special perceptive brain centres of animate creation. The infinite variety of colour seen in external objects depends upon the selective power which such objects possess of absorbing and quenching, in varying degrees, certain of the component spectral colours of the composite light which illuminates them, and reflecting the remainder to the perceptive sense organ.

Associated with every colour sensation the existence of certain distinctive attributes, known as colour constants or elements, can always be distinguished. These are three in number:—(1) *hue*, (2) *purity* or *tint*, and (3) *brightness*, *luminosity*, or *shade*.

Hue is synonymous with ethereal wave-length, for upon this physical factor colour is solely dependent.

Purity or *tint* depends upon the absence of white light; the less this admixture the purer the colour.

Brightness, *luminosity*, or *shade* depends objectively upon the energy of vibrations of the ether particles; and subjectively upon the sensitiveness of our retina to this photogenic physical stimulus.

By means of these varying characteristics or qualities any given colour can be scientifically defined in terms of the constant spectral colours.

Physiological Considerations.—The delicate and complex nervous expansion—the retina—is destined for the amplification and support of the ultimate terminal elements of the optic nerve fibrils—the rods and cones. The phenomena of Purkinje's figures and the blind spot establish the fact that these are the essential visual cells, to which (through the inner layers of the retina) the photogenic ethereal waves must first penetrate before visual impulses can originate.

Upon these nerve terminals the ethereal impulses of from four to eight hundred billions per second—the physical equivalent of their respective colour-sensations—impinge. As to the subsequent modification and qualification they undergo in the remaining retinal layers we are utterly ignorant; but there can be no doubt, from the anatomy of the structure and the relation of the optic tracts to the central nervous system, that the obscure changes which take place in the intervening retinal layers are of great importance to vision, with its subsidiary perceptions of light, colour, and form.

It was formerly supposed that the receptive rods and cones mechanically vibrated in unison with the subtle ethereal undulations; but such rapidity of vibration of organic formed elements is inconceivable, and this direct transference of energy without change of form or quality would appear to be physically impossible. Apart from this consideration, it is (as the Committee of the Royal Society on Colour Vision remarks) "difficult to conceive that matter which is so comparatively gross as the rods and cones which are situated on the retina, can be affected by the merely mechanical action of the vibrations of light." It is obvious, therefore, that some subtle transformation of energy between ethereal vibrations and visual impulses must take place, but the nature of this change is largely a matter of conjecture. In the course of this transformation, the occurrence of certain physical phenomena has been definitely established. Holmgren first recognised the existence of electrical processes; then Boll observed the bleaching of the retinal colour under the influence of light, which Kuehne subsequently demonstrated was the outcome of a chemical change. Again, mechanical alterations as to the distribution of the pigment in the retinal epithelium, and in the shape of the visual rods and cones, have also been observed. These varied forms of transformation of energy are evidently intimately associated with each other in the complex visual act, which involves the transformation of ethereal vibration into the special nervous visual impulse.

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[1] The unit μ equals one-millionth of a millimetre.

Nervous impulses in general are now considered to be the outcome of molecular change, generated (through the medium of some obscure chemical process) as the result of some form of physical stimulus, and propagated along a nerve-fibre. In the special nervous impulse under consideration, the physical stimulus is peculiarly subtle, and the direct transference of ethereal into molecular vibrations is not feasible on physical grounds. Whatever the mode of transformation of light energy into visual impulse may be, it is, as yet, beyond the range of tangible demonstration. Here, however, the "chemical" theory affords a particularly applicable working hypothesis, adherence to which is certainly solicited by the knowledge of the ready decomposition of many chemical substances in the presence of light. This photo-chemical hypothesis assumes the existence of complex unstable and therefore easily-decomposable visual matters in or around the terminal visual cells, which (though not necessarily pigmented) can absorb light, but which do not generate visual impulses until decomposed under its influence. When such decomposition occurs, chemical products are formed which are believed to excite molecular changes in the retinal elements, which changes are transmitted hence up the nerve as the visual impulses of light, colour, and form. In order that the incident light may expend its energy in provoking the necessary intermediary chemical change, it is essential that the photogenic waves be first arrested and absorbed. The minute structure of the retina demonstrates the existence of a specially differentiated brown pigment—fuscin—in the retinal epithelium, which may fulfil this function. The chief pigment of the visual cells—the visual purple or rhodopsin—was, when first discovered in 1876, regarded as the special visual matter, the subject of photo-chemical decomposition. Under the influence of solar light this is first changed to visual yellow (xanthopsin), and subsequently to visual white (leukopsin); while mono-chromatic spectral colours also produce the photo-chemical effect less rapidly in proportion to their absorption by the purple. It was naturally believed that the physical basis of visual impulse and colour-sensation had been isolated; and the fact that the visual efficiency of the rays corresponding to colour is relative to their photo-chemical effect upon the purple, incidentally lent support to this belief. The idea, however, had to be abandoned, for visual purple is only found in the rods, is quite absent in the cones of the human fovea centralis (where vision is most acute), and may be naturally or artificially absent in animals which see well. The visual matters are as yet, therefore, of purely hypothetical existence. Analogy suggests that some such sensitive matters (which are probably colourless and have therefore, so far, escaped detection) *do* exist as the physical basis of visual impulse. These, by varying photo-chemical decomposition under the influence of light of certain wave-lengths, generate corresponding impulses in the retina, which in accordance with their particular characteristics are appreciated by the perceptive visual centres as the relative colour sensations.

The assumption that the retina is the portion of the visual apparatus mainly concerned in the differentiation of normal colour-perception is rendered very probable, and apparently justified by considerations which may be thus enumerated:—

(*a*) *Law of Specific Energy of Nerves.*—Physiological research supports the view that all nerve-impulses (actually in process of transmission) are of identical character, whatever the nature (motor, sensory, or special sensory) of the nerve may be; that the effect produced by the impulse in any given nerve depends, not upon any variation in its structure, but upon its differentiation to a special function, implying a particular peripheral termination on the one hand, and association with a special area of the central nervous system on the other; that the nerve-fibres themselves act as indifferent conductors to the impulses which it is their normal function to transmit; that impulses in any given nerve only generate its own particular effect, whether the stimulus applied be its normal physiological one, or any other (chemical, mechanical, or electrical) capable of exciting it. Thus, a blow on the eye or section of the optic nerve produces a sensation of light; in other words, these crude mechanical stimuli can only be appreciated by the brain as the special sensation which it is in the habit of receiving through this channel.

This phenomenon strongly suggests the probability that the chemical process which initiates the molecular change in nervous impulses is identical in all nerves whatever the nature of the exciting stimulus may be; while the logical outcome of this assumption is the non-tenability of those theories which, by supposing the nerve terminal to undergo different changes according to the colour striking it, endow the nerve-fibre with the faculty for transmission of a variety of nerve-impulse, and make the brain the differentiating organ.

Donders asserts that modern physiology will not countenance such a view, while the researches of Goldscheider in the demonstration of special dermal nerve-endings for the sensory impressions of touch, pain, heat, and cold strengthen, by analogy, the assumption that the retina is the differentiator concerned in colour perception.

In furtherance of the above physiological law, Dr. J. Wallace has tried to demonstrate a definite relation between the length of a cone and the colour to which it responds. This implies an invariable length for each cone, which Stort has proved is not borne out by actual fact.

(b) *Uniocular Colour-blindness.*—Well-authenticated cases of this peculiar congenital condition are known to exist. Its limitation to one eye lends support to the view that the eye rather than the brain is at fault; indeed such cases can only be explained on the assumption of a defect on the peripheral side of the optic chiasma, and probably (as vision is usually of normal acuity) in the retina itself.

Professor Rutherford aptly commented to the effect that were congenital colour-defect of cerebral origin, defective colour-sense on one side of the brain would have implicated half of each eye rather than the whole of one.

The physical and physiological processes involved in colour-perception may be summarised as comprised in three factors, which are essential to the process:—

1. An *external object* with its selective absorptive capacity for colour.
2. The *physical medium* — the ether — transmitting the photogenic vibrations—the specific physiological nerve stimulus.
3. The *receptive subject* with its special sensory apparatus, provided with its specific and highly specialised end-organ. This nervous apparatus is of threefold constitution, comprising:—

(*a*) The *retina* with its chromo-sensitive end-organs, and

(*b*) The *conducting nerve-fibres* for the reception and transmission of the special physical homologous stimulus to

(*c*) The *cerebral centres*, the special perceptive visual organ.

THEORIES OF COLOUR-PERCEPTION.—It is not necessary to assume the existence in us of as many primary colour-sensations as there are colours in the spectrum, for it is found that in order to produce experimentally the multitudinous variety of colour therein seen, only three primary colours are essential. It is therefore generally assumed that our vision is *trichromic*, *i.e.* founded on three, or the equivalent of three, primary colour-sensations. On this assumption the two main theories of colour-perception are founded.

Young-Helmholtz Theory.—This hypothesis, propounded by Thomas Young (1807), and subsequently elaborated by Helmholtz (1852), affords a clear and simple explanation of most of the complex physiological phenomena of colour, and is now universally accepted among physicists. It assumes the existence of three kinds of nerve-fibres identical as to structure and conduction, but terminating in different end-organs endowed with photo-chemical substances respectively sensitive to the three primary colours—red, green, and blue. In every colour-sensation these three primary colour nerve elements are stimulated, though in varying degrees, the resulting colour-sensation being determined by the proportionate amount of the three elementary sensations. In the accompanying coloured plate the relative degree of stimulation required for each of the spectral colours is indicated by the height of the colour curves and by the depth of the colour.

These three symptoms of nerve-fibres are connected with three functionally different systems of cerebral ganglion cells, whose specific energy fits them for the perception of the associated colour-sensation. The retina is thus made the selective or differentiating organ, and the brain the perceptive one.

The minute anatomy of the retina is looked upon as supporting this theory, for the longitudinal striation in the outer segments of the cones is regarded as constituting them multiple terminal end-organs. Further—the cones being the end-organs concerned in the perception of colour (Max Schultze)—the acuteness of colour-perception should be proportionate to their numerical distribution. This presumption is borne out by actual fact, for colour-perception is most acute at the macula which contains only cones, and diminishes relative to distance from this point, while at the periphery of the retina it is quite absent.

Many objections to this theory have been raised. It assumes the existence of a separate nerve-fibre to each terminal element. Salzer found about three million cones in the human retina, while there are only about one million in the optic nerve. Although as to the fovea the assumption is an essential, it is not necessary in the retinal periphery; indeed, Helmholtz explained the imperfections of peripheral vision on the assumption that several nerve elements in this situation have one nerve-fibre in common.

A further objection—originally raised by Fick and recently revived—is founded on the fact that a small pencil of rays from a distant star appears white, implying, according to the theory, that the image falls on three cones simultaneously, whereas astronomical considerations as to distance show that the tiny image cannot cover more than one cone.

This difficulty is ingeniously explained by Thomas and Welland, who suggest that the imperceptibly minute ocular oscillations constantly taking place in accordance with the rhythmical innervation of the ocular muscles, rapidly but successively expose several cones to the incident rays, and that these rapidly succeeding impressions on different colour cells are collectively appreciated by the brain as white light.

The assertion of Charpentier that this theory does not explain the fact of the central retina being less sensitive to colour than to white light is fallacious, for it assumes that while monochromatic light only stimulates one brain-cell, white light excites three equally, so that the sensitiveness of the retina to compound light is proportionately increased.

Other objections against this theory have been urged, but for the present purpose they do not require discussion.

Hering's Theory.—This rival theory—a modification of the Young-Helmholtz—was first propounded about twenty years ago by Hering of Prague. It assumes the existence in the retina of three visual substances—white-black, red-green, and yellow-blue (in each pair one colour being complementary and also antagonistic to the other), corresponding to the supposition that we possess six fundamental sensations. As in the metabolism of living substances generally, the existence of two phases can be recognised—a constructive, assimilative, or anabolic phase, and a destructive, dissimilative, or katabolic phase—so similar metabolic changes of opposite character in the hypothetical visual substances under the influence of light are presumed to be the causal factors in the determination of nervous impulses, and their psychological expression as conscious visual colour-sensations.

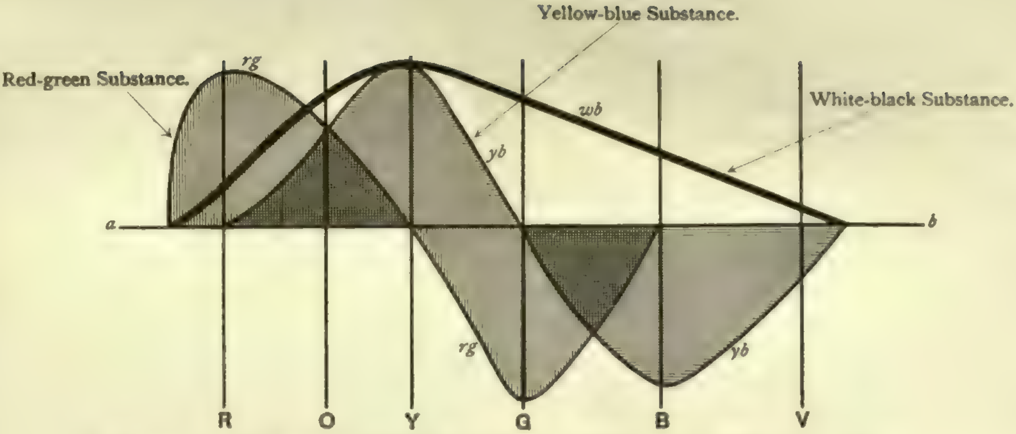


Fig. 1. *DIAGRAM ILLUSTRATING HERING'S THEORY OF COLOUR PERCEPTION.*

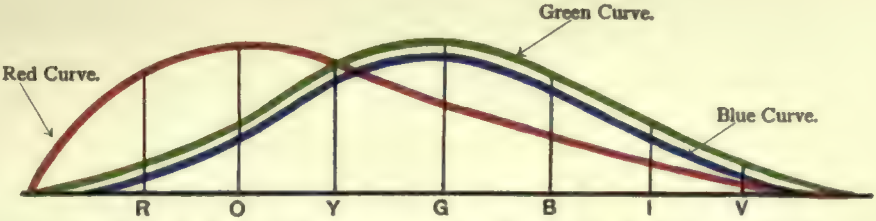


Fig. 2. *BLUE-BLINDNESS* ACCORDING TO THE *YOUNG-HELMHOLTZ THEORY.*

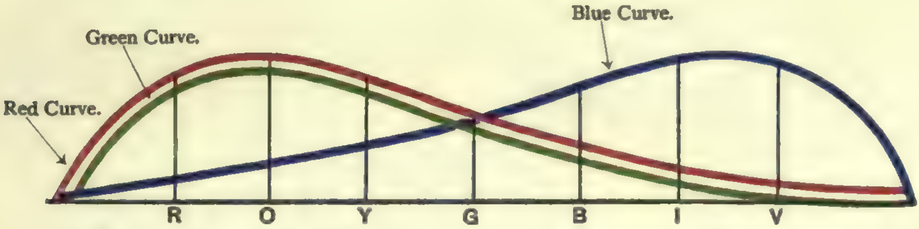


Fig. 3. *GREEN-BLINDNESS* ACCORDING TO THE *YOUNG-HELMHOLTZ THEORY.*

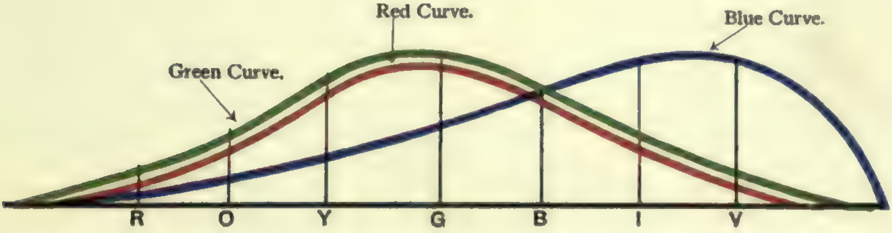
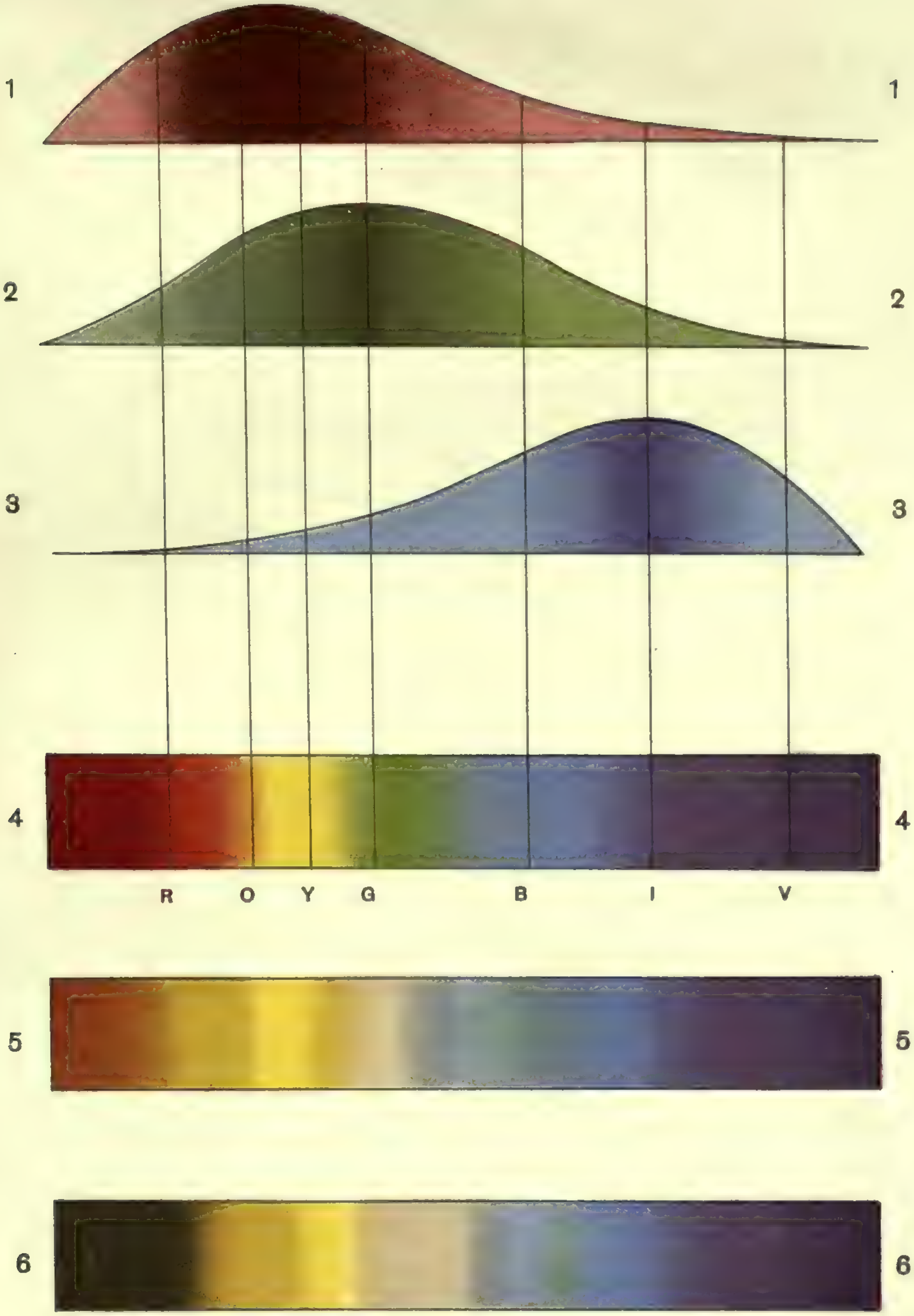


Fig. 4. *RED-BLINDNESS* ACCORDING TO THE *YOUNG-HELMHOLTZ THEORY.*



1, 2, 3, 4 diagrammatically represent the Young-Helmholtz Theory of colour-perception. 1. The red-sensitive substance. 2. The green-sensitive substance. 3. The blue sensitive substance. 4. The solar spectrum of the normal eye. The vertical lines correspond to the spectral colours, and indicate the relative degree of stimulation of the three photo-sensitive substances—the degree being proportional to the height of the colour-curve and the tint of the colour.

5. The solar spectrum of the green-blind person.

6. The solar spectrum of the red-blind person.

Different rays of light, according to their wave-length, affect the several visual substances differently, provoking constructive or destructive changes in some, while others may be left in equilibrium, and producing a resulting colour-sensation expressive of the balance of effect on the whole. The accompanying diagram from Foster's *Text-Book of Physiology* (sixth edition, part iv. p. 95) serves to indicate the nature of Hering's assumption with regard to colour-perception.

The vertical lines R, O, Y, G, B, V indicate the position on the spectrum of the corresponding colour whose initials they bear. The curved line *rg* (enclosing a space shaded vertically) represents the red-green substance, the effect of rays of different wave-length upon it being indicated by the height of the curves. The line *yb* (enclosing a space shaded horizontally) represents the yellow-blue substance, the effect of various rays upon it being indicated in the same manner. The horizontal line *ab* separates the katabolic, dissimilative, or destructive changes (represented above the line) from the anabolic, assimilative, or constructive changes (shown below it). In the red-green substance, as far as the line Y (*i.e.* for red, orange, yellow) the effect is katabolic, producing a red sensation; beyond this point (*i.e.* for green and blue) the effect is anabolic, producing a green sensation. In the yellow-blue substance the effect is katabolic up to G (*i.e.* for orange, yellow, and green), producing the sensation of yellow; but beyond this point (*i.e.* for blue and violet) it is anabolic, producing the sensation of blue. The thick line *wb* (enclosing an unshaded space) indicates the white-black substance which is only affected katabolically, though to a variable degree, by *all* the spectral colours, producing the sensation of white. Its anabolism produces the sensation of darkness.

Red light (R) produces the sensation of red by inducing a katabolic change in the red-green substance. Orange induces katabolic changes in both the red-green and yellow-blue substances. Yellow produces katabolism of the yellow-blue substance, the red-green now being in equilibrium (its curve intersecting the line *ab*). Green induces anabolic changes in the red-green substance, the yellow-blue now being in equilibrium. Blue produces anabolic changes in the yellow-blue substance; violet also does the same, but to a lesser degree. The simultaneous effect of mixed lights is the sum-total of the effect of their several component colours.

Ebbinghaus has modified Hering's theory by admitting only katabolic changes in the three visual substances as the cause of colour-sensation. He attributes colour properties to the visual purple, which latter he considers identical with the yellow-blue substance. He asserts that it exists in the cones as well as in the rods, and that its apparent absence in the former is due to its being colourless from admixture with the complementary red-green substance, which is present only in the cones. The outcome of this assumption, viz. that the nerve of any cone transmits impulses varying according to the colour, is, however, opposed to the views of present-day nerve physiology.

The above two theories are the only hypotheses relating to colour-perception which now hold the field; and though there are still adherents to Hering's theory, that of Young-Helmholtz meets with the more universal acceptance. All other theories which endow the optic nerve-fibres with the faculty of transmitting nerve-impulses of varying characteristics are antagonistic to the accepted facts of present-day nerve physiology.

Development of Colour-perception.—Colour-sense and colour-knowledge must not be confounded; the former is an inherited instinct, the latter results from education and practice of the sense.

Ample evidence exists that the colour-sense was coexistent with primitive man of whom we possess any trace. Among the deposits found in the sepulchral barrows of prehistoric times are variously coloured glass or quartz beads and elaborately artistic implements and pottery; while the enamelled bricks of Nineveh and Babylon and the Egyptian mummy cases show that the ancient inhabitants of the land of the Pharaohs possessed a colour-sense of the highest order.

It is evident to any careful observer that a keen appreciation of colour is one of the marked endowments of early infancy. The colour vocabulary is absent, but education in the nomenclature of colour is alone required to voice the existence of the sense. The ordinary vocabulary for colour is a simple one—black, white, red, blue, green, yellow, gray, brown; and though the majority of educated individuals are content with so meagre a list, there is no reason why such should not be indefinitely extended. Though we have no means of knowing how certain terms were originally used or applied to designate the primary colours, the abstract nature of colour in the development of the colour vocabulary is interesting. Most of our colour-names take their origin from some concrete object. Thus the baking of a certain kind of clay gives us "brick-red," while another variety of clay gives us "terra-cotta." In "rose-colour," "cardinal," "claret," the concrete origin of the colour-name is still apparent. "Rosy," "roseate," "pink," "carnation," "cherry," and "plum" are further examples, while stones such as amethyst, amber, sapphire, turquoise, topaz, and jet give us still more conclusive evidence. Probably the only difference between concrete and abstract colour-names is one of time. Orange, lilac, and even so recent an addition as that of electric-blue, are fast losing association with the objects from which the names were originally derived.

The natural colours with which civilised man is brought into contact are comparatively few; the artificially produced hues are many and increasing. A pattern-card issued by a Lyons silk manufacturer contains samples of two thousand different colours, each with its more or less appropriate name; and while this colour vocabulary is larger than the entire vocabulary of the majority of people, the gradations of colour in the solar spectrum are still more numerous. The sense to perceive these is already existent; as opportunity offers, an extended colour vocabulary will result.

Definition.—Colour-blindness is the condition in which there is inability to distinguish certain colours. It may be *congenital* or *acquired.* The subject may be discussed in terms either of the Young-Helmholtz or Hering's theory.

Congenital Colour-blindness.—*Historical Considerations.*—This congenital defect has probably been coexistent with the human race. It was so named by Brewster. The first undoubted case appears to be one recorded by Huddart in the *Philosophical Transactions* of 1777, which was subsequently commented on in 1779 by the Abbé Rozier, who also referred to the case of the painter Calarean. The same year the case of Scott was reported. Then followed the historic case of Dalton, the English chemist of atomic theory fame, who in 1794 studied his red-blindness by the spectrum, and accurately described the condition. The subject was first systematically and theoretically studied by Seebeck about 1836. George Wilson of Edinburgh (1818-1859), by his researches and writings, strongly advocated attention to the practical side of the question, but his work did not receive the attention it deserved. The measure of attention which the subject in its practical bearings is now receiving is largely due to the powerful advocacy of Frithiof Holmgren of Upsala, in his work published in 1877, and of Dr. B. Joy Jeffries of Boston, who wrote two years later. Yet even now, in spite of constant and consistent agitation, much remains to be done, for Governments do not yet fully recognise their duty in a matter of such vital importance to life and property.

Congenital colour-blindness may be *total* or *partial.*

Total or Complete Colour-blindness—Achromatopsia.—This condition is rare; it may be unilateral (Otto Becker and von Hippel). The spectrum appears in shades of gray throughout, being lightest in the position of the yellow-green, and darkest at each end. A coloured picture appears like a photograph or an engraving. According to the Young-Helmholtz theory, such cases are explicable on the assumption that all the three photo-chemical substances are alike, as is normally the case at the extreme retinal periphery, or that the three colour-receptive elements are paralysed. Hering's theory asserts that all the chromatic substances are absent, the white-black alone remaining.

Mono-chromatopsia.—This is a very rare group akin to the above, in which the whole spectrum appears in shades of some one colour—green or blue-violet. In such cases we assume the existence of the corresponding photo-chemical substance only.

Partial Colour-blindness.—Of this condition there are *three* varieties designated (according to the Young-Helmholtz theory)—blue-blindness, green-blindness, and red-blindness. All are characterised by the spectrum appearing dichromic, with a neutral area of white or gray, as shown in the accompanying diagram.

Blue-blindness, violet-blindness, yellow-blue blindness of Hering, akyanopsia, axanthopsia.—This group is very rare, and therefore not of practical importance. The spectrum consists only of red and green, with a neutral gray area in the yellow; the blue-violet end is usually much shortened. In typical cases only red and green are seen perfectly (Erythrochloropsy of Mauthner), blue is confounded with green, purple with red, orange with yellow, and violet with yellow-green or gray. According to Young-Helmholtz, the blue-sensitive substance is here equal to, or has the properties of the green-sensitive substance, as diagrammatically represented by the parallelism and proximity of the green and blue curves in the figure appended. From this it is evident that at Y (the point of intersection of the curves), where in normal eyes the sensation of yellow would be perceived, all the three substances would be equally stimulated, and the sensation of gray in consequence provoked.

Hering's hypothesis explains the condition by the absence of the yellow-blue substance. Uniocular cases have been recorded by Holmgren and other observers.

Green-blindness, Hering's red-green blindness with unshortened spectrum, achloropsia, aglaukopsia, xanthokyanopy of Mauthner.—This is a group of great practical importance. The unshortened spectrum contains no green, but appears to consist of two colours only, usually called various shades of yellow and blue, which colours join each other directly, or are separated by a narrow neutral gray band. In such cases bright green is confounded with dark-red, and a dark-green letter on a black ground is not recognised.

According to Young-Helmholtz the green-sensitive retinal substance is here equal to, or has the properties of the red-sensitive matter, as indicated in the accompanying diagram by the close proximity of the green to the red curve. From this it is obvious that at G (the point of intersection of the curves), where normally green would be perceived, all the three sensitive substances are stimulated equally, and the sensation of gray is brought about.

Red-blindness, Hering's red-green blindness with shortened spectrum, anerythropsia, Daltonism.—The spectrum is shortened owing to the absence of the red end, and is composed (as in green-blindness) of two colours—yellow and blue. The differences are that the neutral gray zone between the yellow and blue is now nearer the blue end, and that the yellow begins in the orange and not in the red as in green-blindness. In such cases light-red is confounded with dark-green, and a dark-red letter on a black ground is not recognised.

The Young-Helmholtz theory asserts that the red-sensitive substance is here equal to, or has the properties of the green-sensitive substance. This is indicated in the accompanying diagram by the closely adjoining position of the red to the green curve. Here red at R is stimulated so slightly that the sensation is barely provoked, while at the site of intersection of the curves, G B, the normal greenish-blue is perceived as gray, for here all the three sensitive substances are stimulated equally.

According to Hering's theory, green and red-blindness are varieties of red-green blindness due to absence of the red-green visual substance, the difference in response to tests being due to variation in colour of the ocular media—the crystalline lens and the macula lutea. This is hardly feasible, for lens discoloration only occurs in senile or diseased eyes, whilst the macular pigment, which undoubtedly does affect greenish-blue rays, barely influences those concerned in the above difference.

Incomplete Colour-blindness, Diminished Colour-sense, Dyschromatopsia.—In these cases, which are of frequent occurrence, the acuteness of colour-perception is reduced. When objects are sufficiently large and well illuminated, colours are recognised; but when seen under unfavourable conditions as to illumination, or mixed with white or gray, the ground colours are less readily detected than by the normal eye. All the spectral colours are appreciated except that violet cannot be distinguished from blue, or is called gray or brown. Pure colours are easily recognised, but when mixed, difficulty is experienced in picking out the dominant one, and gradations in colour are less obvious.

The acuteness of colour-perception in persons with normal trichromatic vision is not invariably the same; indeed a difference may be noticed in the eyes of the same individual.

These slight aberrations from the normal condition are probably attributable to a reduced sensibility or an alteration in the relation of the three photo-chemical substances, whereby they simulate each other in properties, and manifest their distinctive effects less potently.

Colour-ignorance.—This is ignorance as to colour nomenclature, and not inability to discriminate between colours. Its existence in the adult is very doubtful, as evidenced by the fact that in spite of the institution of a special colour-ignorance test in the British Mercantile Marine Regulations of 1894, no failure to pass this test has hitherto been reported. It only occurs in boys of the lowest social strata whose environment has led to a total neglect of education in the naming of colours.

Influence of Heredity and Sex on Congenital Colour-blindness.—These are important factors in the genesis of this condition. It occurs much more frequently in the male sex, and is usually transmitted from grandfather to grandson, whilst the intermediate generation — both male and female members—frequently escape the defect altogether. Several brothers may be afflicted with the abnormality, in such cases the defect usually assuming the same type in all. Very rarely transference of the defect to the female members of a family is seen. The visual acuity is usually unimpaired. Uniocular cases occur, but exclusively among men.

Prevalence of Congenital Colour-blindness.—Well-authenticated statistics of numerous observers who have collectively examined nearly one hundred thousand persons demonstrate two facts:—

(*a*) Its prevalence among the male sex.

(*b*) Its equal distribution in different nationalities.

These investigations show an average of 3·59 per cent colour-blind among males, and only ·88 per cent among females. This rare occurrence in the female sex has been attributed to their special earlier individual education in colour; while this does undoubtedly account for the absence among them of colour-ignorance, it is more probable that their highly developed colour-sensation is an hereditary endowment peculiar to their sex,—the outcome of a special educational development which has been in progress for ages

Acquired Colour-blindness.—This is a condition of defective colour-perception due to pathological changes, affecting those whose colour vision was formerly unimpaired. In the normal trichromic eye, the visual colour-fields are physiologically limited to certain definite areas, which vary in extent according to the particular colour, and are none of them coextensive with the field for form. Colour-perception is perfect only over a limited eccentric area surrounding the macula. Beyond this limit, deviations normally occur similar to those observed in the colour-blind throughout the whole retina—the differentiation of green, red, and blue being respectively lost, as the retinal periphery is approached, while at the extreme periphery is an achromatic zone capable only of form vision.

The distinctive symptomatic difference between congenital and acquired colour-blindness is the associated failure of visual acuity for form almost always present in the acquired variety. The defect usually annuls or abolishes the colour-perception in a definite sequence — the order of colour-disturbance being green, red, and lastly blue. It is more frequently dependent upon pathological changes affecting the conducting nerve-fibres or the colour-sensitive cerebral cells, than to a lesion of the third factor in colour-preception, viz. the retinal chromo-sensitive visual cells. This ætiological fact is probably explained by the apparently greater resistance to pathological processes offered by the photo-chemical substances than by the optic nerve-fibrils, as evidenced by the observation of Stilling, who found the colour-sense unimpaired in various forms of retinitis and choroido-retinitis, and by the statement of Leber that in such retinal affections colour-blindness only ensues when the disease has led to secondary atrophy of the nerve-fibre layer and of the optic nerve.

The affection assumes a variety of types as to distribution, according to the pathological factor upon which it depends. The main varieties and the associated causes may be thus classified :—

1. The colour defect may involve to a variable extent the retinal circumference only, and remain localised to these peripheral parts. Form vision is correspondingly restricted, the condition known as "telescopic vision" resulting. This condition follows the administration of various therapeutic agents, such as quinine, salicylate of soda, ergot, and caffeine. These drugs, from idiosyncrasy or overdose, produce toxic effects, by disturbing the vaso-motor centres and inducing a condition of retinal anæmia.

2. It may primarily involve the retinal periphery, and progressively spread over the whole retina. This is the most frequent form, occurring concurrently with optic nerve atrophy, whether *primary* and associated with general nervous disease, or *secondary* and consecutive to neuritis from any cause.

3. The defect may be limited to the central portion of the retina only, producing a central colour scotoma, which may be pericentric or paracentric according as it includes the fixation point or not. This variety includes the so-called toxic amblyopias which in reality depend pathologically upon retro-bulbar axial neuritis. The toxic agents producing this condition are alcohol, tobacco, bisulphide of carbon, iodoform, nitro-benzol, cannabis indica, arsenic, lead, stramonium and chloral hydrate. Central colour scotoma are also of frequent occurrence in disseminated sclerosis, and sometimes as the outcome of toxæmia in diabetes.

4. It may involve the corresponding halves of each retina (chromatic hemianopsia) as in some rare cases of cerebral hæmorrhage or embolism—the field for form being unaffected. In ordinary hemiopia the loss of colour corresponds to loss of form.

5. Disturbance of normal colour-perception is of frequent occurrence in certain functional neuroses, as hysteria, epilepsy, and hypnotism. For a detailed account of the special colour phenomena in these and in the above-mentioned groups the reader is referred to the special articles.

Signal Colours.—By universal custom, the colours—red, green, and white—have been adopted as signals to indicate conditions of danger and safety. When the prevalence of colour-blindness in the male sex was duly recognised, it was found that most colour-blind men were either red-blind or green-blind; and the advisability of replacing the above colours by others less readily mistaken was considered. Experiments, however, as to the luminosity of various coloured lights have satisfactorily proved that the colours in usage are the best and the only available signal lights for practical purposes; for, while red transmits 10 per cent, and green 10 to 20 per cent of the light behind it, blue only allows 4 per cent to pass through, while yellow under certain atmospheric conditions would not be sufficiently distinctive. The elimination of the colour-blind from positions of vital responsibility became, therefore, a grave necessity, and upwards of forty methods for the detection have been devised.

METHODS OF TESTING.—Any method to be scientific and conclusive must be based on the principle of the *matching* of colours, since this is the only true appeal to the colour-sense. The *naming* of colours is fallacious, for the truly colour-blind may guess correctly, while from colour-ignorance or nervousness the colour-perfect may fail. In methods involving the matching of colours, coloured pigments seen by reflected or transmitted light or the spectral colours may be employed. Of these the simplest and most practical test is that of Holmgren.

A. *Wool Test of Holmgren.*—Seebeck was the first to mention coloured worsteds, while Wilson of Edinburgh was the first to employ them. Holmgren's method is an elaboration of the suggestion, five finely graduated shades of each of the following colours—red, orange, yellow, greenish-yellow, green, greenish-blue, blue, violet, purple, rose, brown and gray—being employed.

The test consists in requiring the examinee to match from the heap of wools of various colours and shades (confusion colours) placed on a white cloth in a good light "test skeins" of certain definite colours. These test skeins are three in number, and are applied in a given order.

Test 1.—The examinee is given a skein of very pure pale green (which should be held apart) and told to select from the heap all the skeins which contain any tint of that colour, care being taken to avoid specifying the colour of the test skein. If any but green skeins be selected by the examinee as a match, colour-blindness is established; while a marked disposition to select such, though finally not yielded to, indicates a feeble colour-sense. Should the examinee have difficulty in understanding what is required of him, the examiner himself may show him by selecting the required skeins without in any way invalidating the value of the test.

To ascertain the kind and degree of the colour-defect two further tests may be employed:—

Test 2.—A rose-coloured skein (a mixture principally of red and blue in the proportion of two to one) is now given to the examinee, and he is asked to match it. If he select blue and purple skeins he is completely red-blind, while if he select the blue-green skeins he is completely green-blind.

Test 3.—A bright-red skein is now presented. The complete red-blind selects as matches to this, dark green and reds, and browns (*i.e.* shades which to the normal eye appear darker), while the complete green-blind selects light greens and light browns (*i.e.* shades which to the normal sense appear lighter than the test colour).

It does not always follow that the mistakes of the colour-blind are invariably those indicated above. As normal colour-perception varies in different individuals, so in the colour-blind every gradation as to kind and degree of defect exists. While typical cases of either form respond, therefore, in a distinctive manner to the tests employed, incomplete cases will be indicated by deviation from these regular lines.

Various modifications of this method have been devised for facilitating the examination and recording the results, when dealing with large bodies of men. These may be briefly enumerated as follow:—1. Thomson's colour stick; 2. Jeaf-

ferson's colour disc; 3. C. A. Oliver's wool test; 4. Daae's wool squares; 5. Reuss' colour table; 6. Cohn's embroidery patterns; 7. Bodal's coloured cylinders; 8. Schenke's yarn spools; 9. Donders' wool rolls.

B. *Pigment Tests.*—Cohn and Mauthner advocate the use of bottles filled with different pigments in place of worsted.

C. The *chromo-lithographic tables of Stilling* consist of coloured letters on differently coloured backgrounds, seen under reflected light. Grossmann's modification embodies this idea, but the coloured slides are shown by transmitted light.

The above-mentioned methods only permit of a qualitative determination of the colour-sense. To determine this quantitatively recourse must be had to one of several methods devised by Donders, Oliver, and Brudenell Carter.

For a truly scientific estimation of the colour-sense the spectral colours must be employed. Lord Rayleigh, Abney, Chibret, and Hirschberg have devised instruments by means of which definite spectral colours can be exposed and matched for selection from another spectrum.

PRACTICAL REMARKS.—Seamen.—The practical importance of the subject of colour-blindness was not recognised until 1855, when Dr. George Wilson of Edinburgh published his *Researches on Colour-Blindness*, and graphically drew attention to the dangers of employing colour-blind men in positions of trust. In 1876 a railway disaster at Arlsey Junction, and the suggestion that it was due to colour-blindness on the part of the engine-driver, excited public attention. In the following year the Board of Trade established a compulsory test for those of the Mercantile Marine applying for certificates of master and mate. The test consisted in the *naming* of colours, the consequence being that men rejected at one time or place passed the test successfully on another occasion. The perfunctory manner in which the Regulations respecting colour-blind men were drawn is shown by the following facts:—

1. That no test was required of "look-outs," ordinary seamen, apprentices, or pilots.
2. That the colour-blind officer received his certificate, though it was endorsed "colour-blind."
3. That no provision was made for colour-blind officers who might conscientiously feel their unfitness for the sea life.

The inefficiency of the so-called "colour-blind test" led to a long controversy between members of the medical profession and the Board of Trade, resulting finally in the establishment of the Holmgren Wool Test as the official test on September 1, 1894. The Board have not, however, yet risen to a full sense of their responsibility. The primary object of Government is to provide safeguards for human life which individual care cannot command. From this standpoint their own Reports condemn them. Between 1877 and 1894, 72,894 sailors already holding or applying for certificates were compulsorily examined, 409 failed to pass in colour. In the same period, 6370 *voluntarily* offered themselves for examination, and 293 failed to pass. Since 1894, when the Holmgren test came into vogue, 21,720 have been tested, with 232 failures, reduced on appeal to 215. Taking the figures of both periods together, between 1877 and 1898, 100,984 sailors have been tested, and 917 found colour-blind. Of these 917 colour-blinds there is not one tittle of evidence to show that they have abandoned the sea life; on the contrary, we know that in spite of their defect they are allowed to continue their calling, and do so to the menace of the public safety. The obvious deduction is that while the Board of Trade completely fail to procure that measure of protection which the public requirements demand, the public on their part have, by the adoption of fallacious Regulations, been deluded into a state of false security. The recommendations of the Royal Society's Committee that *all* candidates for positions of trust should be compulsorily examined, and that in judicial inquiries as to collisions the witnesses should be examined for colour-blindness, would, if carried out, open an era of safety which has hitherto been unknown.

Railway Men.—Up to the present time there has been no agreement among the various railway companies as to the application of a recognised standard colour-test. The consequence is that colour-blind men may still be found in the ranks of railway employees. Recent agitation on the part of the British Medical Association will no doubt result finally in the universal adoption of Holmgren's test, supplemented if necessary by some quantitative method.

Comedones. *See* Acne, Sebaceous Glands.

Concussion. *See* Brain, Intracranial Surgery.

Condyloma. *See* Syphilis.

Congenital Dislocations. *See* Deformities.

Conjunctiva, Diseases of.

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Anatomy of the Conjunctiva

The conjunctiva externally is continuous with the skin at the free edge of the eyelids, and internally with the Schneiderian membrane by way of the lacrymal ducts. The pinkish *palpebral conjunctiva* is firmly attached to the inner surface of the eyelids, and is connected with the ocular conjunctiva by the retro-tarsal folds, one above and one below, which by their loose arrangement prevent any dragging upon the lids by the movements of the eyeball. The *ocular conjunctiva* lies upon the anterior part of the sclera; it is pale and thin enough to allow the sclera to be seen through its texture. It is loosely attached to the underlying sclerotic, so that it may be readily picked up with forceps. By a sort of reduplication it forms towards the inner angle of the eyelids a more or less vertical fold, the plica semilunaris, a vestigial relic of the third eyelid of certain lower animals. The small reddish mass, often covered with fine hairs, which lies between the plica and the inner commissure of the eyelids, is called the caruncle. Around the cornea the ocular conjunctiva becomes closely knit to the underlying sclera, and forms what is known as the *limbus conjunctivæ*, a position in which blood-vessels are especially numerous. The conjunctiva covers the anterior surface of the cornea, but in that position it is reduced to a few layers of epithelial cells. The *nervous supply* of the conjunctiva comes from branches of the fifth cranial nerve; the *vascular supply* mainly from the posterior ciliary vessels. The *lymphatics* comprise a superficial and a deep set, with communicating branches.

MALFORMATIONS AND CONGENITAL ANOMALIES

Fibro-fatty Tumour.—SYN. *Subconjunctival Lipoma or Lipo-dermoid.*—This congenital anomaly is not uncommon, but as it generally causes no marked deformity, it falls comparatively seldom under the notice of the surgeon. The tumour takes the form of a soft, slightly raised yellowish mass, more or less triangular in form, and usually occupying the interval between the superior and the external rectus muscle. It lies beneath the ocular conjunctiva, and is freely movable upon the globe. As a rule, its investing conjunctiva appears normal, but short hairs may be present, a condition to which the name *trichosis bulbi* has been applied. There are a few cases recorded where a tumour occupying the customary position of a fibro-fatty growth was found to contain an osseous nucleus, in some instances covered with periosteum. I believe, as a result of microscopic investigations of these cases, that they are probably of a dermoid nature, and when they contain bone should be classed as teratoma.

Treatment.—When inconspicuous these growths are best left alone. Otherwise, the conjunctiva may be reflected, and some of the exposed fatty-looking material snipped away with fine curved scissors. The conjunctiva is afterwards replaced, and kept in position by inserting a continuous suture.

Dermoid Tumours.—The ordinary dermoid forms a solid, pinkish white growth, usually situated at the lower and outer part of the sclero-corneal junction, and encroaching to a variable extent upon both cornea and conjunctiva. It is attached firmly to the cornea. Hairs may or may not grow from the dermoid, the top of which is now and then dry and fatty-looking. The tumour (generally limited to a single eye) is usually about the size of a split pea. A case has been reported, nevertheless, where the growth had reached the bulk of a horse-bean, while upwards of twelve long hairs grew from its middle part, passed between the eyelids, and hung upon the cheek. The patient remarked that these hairs did not appear until he was sixteen years of age, at which time also his beard grew. The dermoid often shows a distinct tendency to get larger, and to become hairy at or about puberty. The microscope shows that these growths include the elements of ordinary skin, as epithelium, glands, hair follicles, etc.

Treatment.—The growth may be removed by careful dissection.

Nævi.—Angiomata, plexiform or cavernous, may involve the conjunctiva, and are usually associated with a similar condition of the eyelids, orbit, face, or of other parts of the body. The commonest clinical appearance is that of a small or large patch of purplish-red discoloration affecting the palpebral mucous membrane. More rarely a definite tumour is met with, involving, it may be, the semilunar fold or the ocular or the palpebral conjunctiva. In some of these cases the angioma forms a livid, nodulated mass, bleeding on slight provocation.

Treatment.—Small capillary nævi, which give rise to no disfigurement, call for no special treatment unless they show a tendency to get bigger. More conspicuous patches may be seared with the galvano-cautery, or (after carefully drying the parts) be painted lightly with solution of sodium ethylate (P.B.) until improvement results. The cavernous angiomata are best dissected out, an operation that is by no means so difficult as it seems.

Lymphatic varix is a rare but interesting congenital condition which depends upon dilatation of the lymphatic vessels, with hyperplasia of the conjunctiva. Some part of the ocular mucous membrane is occupied by a bunch of small, yellowish elevations, which convey the notion that they contain fluid. The mass may undergo variations in size, particularly in females at the menstrual period. In a case of my own the appearance reminded one of a non-inflammatory chemosis, of yellowish colour, occupying the ocular conjunctiva on the nasal side of the eyeball. In another case a prominent mass of yellow-like tissue, dotted over with tiny hæmorrhagic points, was connected with the semilunar fold, while in the same eye there was a collection of yellowish beaded vessels in the upper part of the ocular conjunctiva. Both lids were thickened, presumably from lymphatic obstruction. The eyeball was small; shreds of persistent pupillary membrane were present; dotted opacities were diffused through the crystalline lens; and sight was extremely poor. Alt examined microscopically an instance of lymphatic varix, and found in the ocular conjunctiva a series of canals and cavities, the walls of which were lined by endothelium and formed by the compressed fibres of the conjunctival tissue.

Moles have been described as existing upon the ocular conjunctiva.

Supernumerary Caruncle.—It appears that the caruncula lacrymalis, like the tragus or the nipple, may be duplicated. The first observation of the kind was published by the writer in 1896, and since then Dr. J. W. H. Eyre has recorded two similar cases. A small, raised, fleshy mass, of granular appearance, is attached to the conjunctiva of upper or lower lid somewhere near the normal caruncle; fine hairs may be present on the surface of the growth.

Treatment.—Should it be desired for æsthetic reasons, the little tumour may readily be snipped away with scissors. Recurrence is unlikely.

Other Rare Affections.—The writer has seen perhaps half-a-dozen instances of a peculiar congenital growth, which, so far as he is aware, has not been mentioned in literature. A grayish red tumour, oblong or reniform in outline, occupies some part of the ocular conjunctiva. It is freely movable with the conjunctiva in which it lies; a few dilated vessels often run towards it. Microscopically it seems to be of cystic nature, its wall being formed of a thick but irregular stratified epithelium, together with a vascular subepithelial layer of connective tissue. The tumour is easily dissected out, and if the wound be carefully closed with a continuous suture, scarcely a trace of the operation remains.

INFLAMMATORY AFFECTIONS

The conjunctival sac, even in a state of health, is seldom altogether free from micro-organisms, of which by far the commonest is the so-called xerosis bacillus; the staphylococcus pyogenes albus (or the staphylococcus epidermidis albus) is also often found. Certain other microbes are present under morbid conditions, as, for example, gonococci, Weeks' bacilli, pneumococci, diplobacilli, bacillus coli communis, diphtheria bacilli, and various pyogenic cocci. They give rise to definite conjunctivitis, the diagnosis of which is nowadays made chiefly from a bacteriological examination. Although our knowledge of the parasitic nature of acute ophthalmia is fairly complete, the same cannot yet be said of the chronic forms, especially of trachoma.

ACUTE OPHTHALMIA

As stated above, in our present state of knowledge it is possible to ascribe nearly every known form of acute ophthalmia to the action of specific microbes. At the same time, to avoid confusion, we may retain the classical clinical divisions and speak of: 1. Muco-purulent ophthalmia; 2. Purulent ophthalmia; 3. Granular ophthalmia; and 4. Membranous ophthalmia.

1. MUCO-PURULENT OPHTHALMIA.—This common affection tends to become epidemic in spring and summer, and at times it is extremely contagious. No age is exempt, although it is, above all things, a childish ailment.

Etiology.—It is associated with several distinct micro-organisms, of which two are more widely spread than the rest put together, namely (*a*) the short and slender bacillus described by Koch and Weeks, and (*b*) the diplobacillus to which attention was directed by Morax a few years ago. In some other countries, however, it would appear to be more generally due to pneumococci. It is sometimes set up by the bacillus coli communis, as well as by the various pyogenic cocci, as the staphylococcus aureus, citreus, and albus, although in the case of the last-named a special predisposing condition of conjunctiva is probably essential to the ophthalmia. I have met with one case (associated with an evil-smelling discharge) where the bacillus pyogenes fœtidus appeared to be the cause. But, on the whole, we may with tolerable confidence say that a severe muco-purulent conjunctivitis in this country is likely to be due to Weeks' bacillus, whereas a much milder subacute form is probably associated with the diplobacillus of Morax. In my experience, the other organisms named above are comparatively rare as causes of conjunctivitis.

Characters.—The upper lids are reddish and puffed; the ocular conjunctiva is bloodshot and perhaps ecchymosed; the palpebral conjunctiva is uniformly red and rather swollen, and its so-called "follicles" (see page 357) are often peculiarly prominent. Yellowish discharge is found about the eyelashes, at the inner canthus, and, mixed with lacrymal fluid, in the recesses of the conjunctiva. Chemosis is seldom a marked feature. Phlyctenulæ about the conjunctiva or elsewhere may be present. The patient complains of his eyes feeling hot and heavy, as well as of a sensation which he often likens to sand or dust in the eye; a degree of photophobia is not rare. In uncomplicated cases the cornea is clear, the iris bright, and the pupil mobile. Speaking generally, there is little tendency to corneal mischief, especially in children. When the inflammation is caused by the pneumococcus there is apt to be a thin, fibrinous deposit upon the conjunctiva of the upper lid. The special sign of diplobacillary inflammation (in addition to its subacute character) is angular blepharitis—that is to say, an excoriation and soreness of the inner and outer canthus. Pus infections are observed in children suffering from a coincident impetigo or discharge from the ear. The bacillus coli sets up an acute conjunctivitis which has no distinguishing feature beyond its tendency to subside spontaneously.

Treatment.—The principles are:—first, to kill the pathogenic organisms by the local application of antiseptics; secondly, to remove morbid secretions; and, thirdly, to relieve special symptoms, such as pain. The best remedy for all severe cases is a 2 per cent solution of silver nitrate applied to the exposed conjunctiva once a day. The acuter symptoms seldom withstand more than three or four applications, but the remedy should be persevered with until microscopical examination shows discharge to be free from the specific organisms. The same agent succeeds in most of the remaining acute muco-purulent inflammations of the conjunctiva, except possibly in that due to the diplobacillus. The latter is best treated by means of a strong solution of zinc sulphate (grs. 10 to the ounce) dropped over the conjunctiva three or four times a day. Chloride of zinc (grs. 5 to the ounce) may also be used with success. For removing discharge from the eye, a saturated solution of boric acid or corrosive sublimate (1:5000) succeeds admirably. The liquid should be applied at or about body temperature, and the lids, whenever possible, should be everted. Pain, when at all severe, may be relieved by the local use of a 1 per cent solution of cocaine hydrochloride, dropped into the eyes at intervals of a few hours. In severe cases it may be necessary to give bromide of potassium or chloral internally.

2. PURULENT OPHTHALMIA.—This ailment results from the gonococcus reaching the conjunctival sac. It is extremely contagious. *Pathologically,* it is characterised by dense cellular infiltration of the subepithelial tissue, and by great thickening of the surface epithelium, in which, by suitable means of hardening and staining, active karymitosis can be seen. The gonococci do not penetrate the mucosa much deeper than the epithelium, most of which is sooner or later shed. The following points may be noted with regard to gonococci and their relationship to cases of purulent ophthalmia:—(*a*) They are scanty to begin with, but become numerous when the discharge gets profuse; (*b*) the thicker the secretion the greater their number; (*c*) they are not seldom mixed with xerosis bacilli and with pus organisms; (*d*) they may persist for several weeks. In practice purulent ophthalmia is met with under two forms, as it affects (1) newly-born children, and (2)

older subjects. The clinical differences between these varieties render a separate description desirable.

(1) OPHTHALMIA NEONATORUM.—This usually develops on the second or third day after birth. Earlier cases are attributable to a lingering labour in which the infant has been infected during the act of parturition, and later ones to inoculation from contaminated lochia. Material in the maternal passages, containing gonococci, clings to the baby's lashes, and is carried into the eye after birth, either by the blinking of the infant, or by the sponges, water, or other articles employed in the first bath. Its leading feature is a discharge, at first thin and whey-like, and later resembling pus. As the case advances the eyelids are greatly thickened by serous effusion, and when thus swollen they tend to become everted when the baby cries. The palpebral conjunctiva (often difficult to expose) is red, thick, folded, and has a villous look "like a finely-injected fœtal stomach" (J. C. Saunders). Chemosis, however, is not common. In this dangerous affection the cornea may be rapidly involved, particularly in premature or syphilitic infants; an abnormal tightness of the eyelids also is unfavourable to recovery. The baby, in marked cases, is often fretful, while diarrhœa is apt to come on, especially if the infant is bottle-fed. The complications include swelling of the preauricular glands, abscesses about the eyelids, purulent discharges from the external genitals of female children, arthritis, and (rarely) inflammation of the serous membranes. Common results are cicatricial changes in the conjunctiva, opacities of the cornea with or without inclusion of the iris, anterior capsular cataract, nystagmus, and squint.[1]

Treatment.—Discharge must be removed frequently by means of antiseptic lotion, as corrosive sublimate (1 : 5000), mercuric cyanide (1 : 1000), potassium permanganate (1 : 1000), naphthol (1 : 5000), boric acid (saturated solution), or chlorine water. The application should be warm, and applied to the everted conjunctiva with a morsel of absorbent cotton wool. A syringe should not be used for the purpose, since it is capable of doing mischief to both patient and operator. In bad cases, during the height of the attack, the baby must be tended by a nurse day and night, as cure depends chiefly upon the care with which the eyes are kept clean. Iced applications to the eyelids find favour in some quarters, but they call for much attention, tend to confine pus, and are difficult to keep in place. On the other hand, the palpebral conjunctiva may be painted with a 2 per cent solution of silver nitrate irrespective of the stage of the disease. The solid silver stick, whether mitigated or otherwise, cannot be recommended, as its use is almost certain to be followed by ulceration of the conjunctiva. The silver solution may be applied once a day, or even twice in severe cases. There are two other remedies that may be substituted for silver nitrate, namely, protargol (50 per cent) and largin (5-10 per cent), both of which are synthetic compounds of silver and proteid substances. They are used in the same way as the silver nitrate—that is, upon lids everted and carefully freed from discharge. The use of one or other of the foregoing preparations should be continued until the discharge gets thin and scanty, and is found no longer to include gonococci. The remedy is then to be employed twice or thrice a week for a longer term, so as to avoid all risks of relapse. If the cornea be hazy when the case comes under notice, physostigmine should be dropped into the eye three or four times a day:—Physostigmin. sulph., grs. 2; cocain. hydrochlor., grs. 5; distilled water, 1 ounce. Atropine sulphate (grs. 2 to the ounce) may be used instead of physostigmine when the cornea is actually ulcerated. Should the ulcer be deep, or have yellow edges, or should there be pus in the anterior chamber, then more vigorous measures must be taken without further delay. For example, an anæsthetic should be given, and the ulcer either freely seared with the galvano-cautery or else thoroughly touched with the liquefied carbolic acid (B.P.) These operations may have to be repeated.

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[1] It must be borne in mind that forms of ophthalmia other than those caused by gonococci are met with in babies. The writer has found gonococci in 66 per cent of his cases; in the others, Weeks' bacilli, diplobacilli, pneumococci, or bacillus coli communis appeared to be the cause of the inflammation.

(2) Purulent Ophthalmia in Older Subjects.—This affection differs from ophthalmia neonatorum in the following respects:—(i.) It is generally confined to a single eye; (ii.) it affects men more often than women; (iii.) its prognosis, especially in persons over twenty years of age, is very grave. Its most frequent cause is auto-inoculation from a coexisting gonorrhœa. The virus is spread by fingers, washing materials, bed linen, or contaminated instruments or dressings; in hot countries flies appear to be the most usual agents of infection.

Symptoms.—After an incubation period averaging about two days, the disease declares itself by pain, photophobia, swelling of the lids, and the discharge of thin, whey-like material from the conjunctiva. The early signs are so little characteristic in the absence of a clear history of inoculation that the case may be readily mistaken for one of catarrhal inflammation, unless the secretion be examined with the microscope and found to contain gonococci. But before long the conjunctiva of the eyelids becomes markedly swollen, villous, and of a deep-red hue; that of the eyeball is infiltrated with serum, so as to form a mound of jelly-like thickening around the cornea (chemosis). Lancinating pains are common, and the eye may be extremely tender. Meanwhile the abundant discharge is thick and yellow, and includes innumerable gonococci. After a few days the swelling of the lids and conjunctiva becomes less marked, and in most cases the condition slowly recedes. The process, however, when severe, almost always sets up more or less conjunctival scarring. The cornea may suffer either early or late. The earlier and much more dangerous ulcerations are to be suspected when there is much chemosis, but are generally discovered only when the swelling of the eyelids has begun to go down. A dulness is observed either in the interpalpebral zone or else in the central region of the cornea; and, while in a few instances the process may go no farther, usually the grayish area becomes converted into an actual ulcer. The latter may be clear or have a yellowish look, in which event it is likely to perforate the cornea, a staphyloma resulting. The later ulcerations may be central or peripheral, but suitable means will generally prevent their spread, especially when they show any tendency to vascularity. The affection, like ophthalmia neonatorum, may be associated with, or followed by, inflammation of the joints, as in a case related by Weiss and Klingelhöffer. Indeed, there is no reason why the other complications named on a former page should not also occur.

Treatment.—In principle, the treatment of ordinary gonorrhœal ophthalmia does not differ from that of the same affection in babies. The results, unhappily, are nothing like so favourable. The patient must be put to bed, and the pain relieved by sedatives or narcotics. During the earlier stages iced compresses of corrosive sublimate lotion (1:5000) may be applied to the swollen lids. Discharge must be removed with one or other of the weak antiseptic lotions mentioned. The plan of irrigating the conjunctival sac with large quantities of a diluted lotion of permanganate lotion with a special irrigator deserves a trial. Gonococci must be destroyed by the application, once or twice a day, of silver nitrate (2 per cent), protargol (50 per cent), or largin (5-10 per cent). Those remedies, however, must be used with great caution until the discharge assumes the characters of pus. Corneal complications must be treated with atropine drops (grs. 2 to the ounce), or with the galvano-cautery, as described elsewhere. Should a "granular" state of the conjunctiva succeed the acute disorder, the sulphate of copper stick, applied daily, will be found the best remedy. When one eye alone is affected an attempt must be made to save the other from invasion. The most satisfactory plan may be briefly described as follows:—A watch-glass crystal is enclosed between two pieces of adhesive plaster, in which holes have been cut. This contrivance (introduced by Dr. Buller) is then fastened in front of the sound eye, which can by those means be inspected at intervals without the risk of its being touched by fingers contaminated with gonococci. As a further safeguard, the patient, as far as may be, should lie upon his affected side.

3. ACUTE GRANULAR OPHTHALMIA.—Granular ophthalmia or trachoma (*v.* p. 358) is essentially a chronic affection, subject to acute exacerbations. The clinical appearances *in acute cases* vary much, but one feature is common to all, namely, the development in the palpebral conjunctiva of many "sago-grain" granulations. A bacteriological investigation of the secretion has in about two-thirds of my cases revealed the existence of an organism indistinguishable from Weeks' bacillus, and this is doubtless the microbe recently described by Dr. Leopold Müller as the specific cause of the malady. Its presence may be explained by an outbreak due to Weeks' bacillus occurring in a community where trachoma was prevalent. A mixed infection is thus set up, which manifests the clinical picture of an acute or subacute trachoma. The disease is invariably followed by chronic trachoma.

Treatment is that of muco-purulent ophthalmia, and, later, when chronic, that of trachoma. It must never be forgotten that acute trachoma is one of the most contagious inflammations of the eye, so that early isolation is of vital importance.

4. MEMBRANOUS OPHTHALMIA.—Several distinct forms of ophthalmia may be associated with a membrane upon the conjunctiva. The Klebs-Löffler bacillus may give rise to a slight or to a severe form of inflammation under conditions that are as yet obscure. The milder form has for years been known as *croupous*, and the more severe and less frequent as *diphtheritic ophthalmia.* The two affections are more frequent in young children, and tend to follow closely zymotic ailments, such as measles and scarlet fever.

(1) *Croupous ophthalmia* is not unlike a rather severe form of muco-purulent conjunctivitis set up by Weeks' bacillus. The lids, often a good deal swollen, can be readily everted, and strings of glutinous secretion frequently stretch from one lid to the other. The ocular conjunctiva is congested, and may be slightly thickened. The striking feature of the affection, however, lies in the gray membranous exudation upon the palpebral and (more rarely) upon the ocular conjunctiva. The membrane can be stripped away, exposing beneath a thickened, red, and bleeding surface. There may be spots of diphtheritis about the lids, nose, or face; the preauricular and angular glands may be involved; the general health is usually good. The disease is not followed by symblepharon or by cicatricial changes in the conjunctiva. The prognosis is favourable. Croupous ophthalmia is comparatively common in London.

(2) *Diphtheritic conjunctivitis* has a clinical appearance so characteristic that a diagnosis can often be made at sight. The discharge, at first thin and scanty, after a few days turns to a thinnish pus, in which lie shreds of necrotic tissue. If the swollen lids can be everted, the palpebral conjunctiva will be found firm and larda-

ceous, or "brawny," either throughout or in patches. A marked feature is the presence of depressed, grayish-white areas, associated with small, dark-red, ecchymotic spots. The bulbar conjunctiva and the cornea may be covered with false membrane. The affection is followed by cicatricial changes and deformities of the lids. Diphtheria of the fauces or elsewhere may precede, accompany, or follow the ophthalmia. The patients, who are seriously ill, show such symptoms as raised temperature, frequent pulse, depression, anæmia, albumin in the urine, and loss of knee-jerks. A sequel is peripheral neuritis, as shown by paresis of the palate, ocular muscles, extremities, and so on. The disease is very rare in England.

Treatment.—If the clinical evidence of diphtheria is strong, antitoxin should be used without waiting for the results of a bacteriological examination: otherwise the injection may be deferred for a time. Where Klebs-Löffler bacilli are found, antitoxin should be immediately administered. The effect of the remedy, when used early, is remarkable. Experience has proved that antitoxin alone effects a cure. It is advisable, however, to employ local treatment as well—first, to hinder the absorption of toxins; and, secondly, to destroy organisms, other than Klebs-Löffler, that may be present. For this purpose, in the earlier stages, the writer prefers a 15 per cent solution of potassium permanganate, and, later, a 2 per cent solution of silver nitrate. The remedy selected must be applied once or twice daily, if possible, to the everted conjunctiva. Meanwhile the eyes must be kept clean with an antiseptic lotion, such as corrosive sublimate (1:5000), or boric acid (1 per cent), or quinine. A good formula is quinine hydrochloride, grs. 2; distilled water, 1 ounce. Stimulants are often needed in severe cases. The patient must be carefully isolated, especially from other children.

CHRONIC OPHTHALMIA

Three forms of conjunctivitis are included under the general description chronic ophthalmia, but before describing them, a few words must be said with regard to the healthy palpebral conjunctiva. The lower tarsal membrane is generally traversed by arborescent vessels, and shows, especially towards its outer side, a number of minute, transparent elevations, the so-called "follicles," which are neither more nor less than collections of small round cells. Another common appearance consistent with health is that of a single row of minute elevations, which lie close to, and parallel with, the free edge of the outer third of the eyelid. The upper tarsal conjunctiva may be smooth or slightly velvety, particularly along its lower convex edge. It is of so thin a texture as often to allow the underlying Meibomian glands to be recognised as so many vertical markings. It is quite common for a few tiny elevations to lie at the corners of the upper tarsal conjunctiva. Deeply-seated vessels may be seen shining through the loose superior retro-tarsal folds. "Follicles" are frequently to be distinguished, and the lobules of the inferior lacrymal (or palpebral) gland can always be found embedded in the outer part of the folds. It is most important to study the characters of the normal conjunctiva attentively, for a want of that knowledge is likely to lead to errors both of diagnosis and of treatment.

1. Chronic Muco-purulent Conjunctivitis.—This affection results in most cases from a neglected acute catarrh. Its subjective symptoms include heaviness and dryness of the eyes at night, frequent blinking, and sensations like those of foreign substances in the eye. Indeed, they are often like those of an uncorrected error of refraction. The palpebral conjunctiva may be unduly red; secretion may be present after sleep; and a common appearance is that of a whitish, frothy spume collected in the corners of the eye. The patients frequently complain of colours around artificial lights, the result of

particles of mucus on the cornea. Complications—as, for example, troublesome lacrymation, blepharitis, and ulcerative keratitis—are more common in elderly subjects.

Treatment.—Wind, smoke, dust, or impure air must be avoided. Errors of refraction or muscular anomalies should be corrected with suitable glasses. The lacrymal passages and the nose must be carefully examined. Weak solutions of silver nitrate (gr. ½-1 to the ounce) often render yeoman service, although they must not be employed for too long, owing to the risks of staining the conjunctiva. Other useful local astringents are zinc sulphate (grs. 1-2), alum (grs. 2), boric acid (grs. 10). In many cases it is advisable to add to the foregoing lotions a little tincture of opium. A remedy in much favour is the yellow lotion[1] of the Austrian Pharmacopœia, dropped into the eye twice or thrice a day. In children by far the best remedy is the familiar yellow oxide of mercury ointment (grs. 10-20). An ointment of copper sulphate, containing ¼ to 2 grs. of the precipitated salt to half an ounce of soft paraffin and an equal amount of hydrous wool fat, is often efficacious. In difficult cases the everted conjunctiva may be touched occasionally with solid alum or bluestone.

2. FOLLICULAR CONJUNCTIVITIS.—The symptoms of this affection resemble those of chronic muco-purulent conjunctivitis, but in addition the "follicles" are unusually prominent. These are rounded or oval, seldom exceed 1·5 mm. in diameter, are more or less transparent, and are generally arranged in rows "like the beads of a rosary." The conjunctiva is rarely deeply involved, and the changes are always more marked in the lower lids than elsewhere. The condition is never associated with consecutive corneal changes. Its predisposing cause appears to lie in the adenoid tendency of certain subjects; its exciting cause may be either general, as insanitary surroundings, or local, as an attack of acute ophthalmia, the prolonged use of atropine or physostigmine, and so on. The best name for this widely-spread condition seems to be *simple folliculitis.*

Treatment.—If the "follicles" give rise to no symptom, and are not associated with discharge from the eye, they are best left alone. Otherwise, the same remedies may be employed as described in the last section. Tannate of lead ointment (10 per cent) often acts well. If the overgrowths be large, there can be no objection to getting rid of them by squeezing, cutting, or other surgical means. A change of air is sometimes beneficial.

3. TRACHOMA.—This important affection is found almost exclusively amongst the poorest classes, and in schools attended by those classes. It may cling to a particular building for years, simply because proper steps are not taken to eradicate it. Certain races—as Russians, Poles, Armenians, Jews, and Irish—are peculiarly subject to the disease, probably because their mode of life favours the spread of infection. Negroes, curiously enough, are said to be practically exempt. In some countries, as Switzerland, trachoma is almost unknown; in others, as Belgium, it is widely spread. It arises only by infection, but we know nothing definite as regards the pathogenic agent, thought by some to be a micro-organism, and by others to be a protozoon. This much, however, is certain—that infection is fostered by a gregarious life, by bad ventilation, by lack of cleanliness, by defective washing arrangements, and by the absence of isolation for those affected. Indeed, the clinical evidence of the

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[1] The yellow lotion contains ammonium chloride, 0·5, and zinc sulphate, 1·25 parts, dissolved in 200 parts of distilled water. To that solution are added a mixture of absolute alcohol 20 parts, camphor 0·4, and saffron 0·1. The two solutions are mixed, allowed to stand for twenty-four hours, and filtered.

transmissibility of trachoma is overwhelming. Both eyes are usually affected sooner or later.

Symptoms.—Trachoma is essentially a chronic ailment, subject (particularly in the earlier stages) to acute or subacute exacerbations. To begin with, it affects the palpebral conjunctiva, but, later, may spread to the cornea. Its leading features are—first, the development of "sago-grain" bodies in the conjunctiva; and, secondly, thickening of the palpebral mucous membrane. The "sago-grain body," which is almost essential to diagnosis, appears to be a "follicle," altered as the result of specific invasion, presumably by a micro-parasite. It is a round, opaque, grayish-white growth, deeply embedded in the hypertrophied conjunctiva, especially of the upper cul-de-sac. Microscopically, it consists of grouped lymphocytes, supported by a delicate stroma, and often more or less distinctly encapsulated. According to some recent researches, it may originate in the lymph-vessels or spaces of the conjunctiva. Discharge, except in acute cases, is seldom abundant. An almost constant sign of trachoma is ptosis, which gives the patient a sleepy look. The cornea may suffer in two ways, namely, by the development of pannus or of ulceration. A well-marked pannus gives a characteristic appearance to the upper third or so of the membrane. The affected parts are irregular and cloudy, and few or many enlarged vessels (continuous with those of the conjunctiva) branch out into the web. In bad cases the entire cornea may suffer, and vision be reduced to bare perception of light. Indolent ulceration is not uncommon along the free edge of a pannus. The usual explanation of the origin of pannus assumes that it is due to the irritation of a rough and thickened upper lid. This theory, nevertheless, is hardly borne out by the known facts. Without entering into contentious points, it may be safely asserted that pannus represents an actual trachoma of the cornea, brought about either by direct inoculation from the affected lid, or else by the metastasis of infective particles through vessels or lymphatics. Ulcerations of the cornea simply mean disintegration of the diseased material. The sequels of trachoma include scarring of the palpebral conjunctiva, shortening of the culs-de-sac, trichiasis, entropion, and imperfect sight as the result of corneal blemishes.

Treatment.—*Escharotic*, *surgical*, and *accessory means* are used. For general use, the best *escharotic* is solid bluestone, rubbed over the exposed palpebral conjunctiva once a day. The application may be continued for years without harm. Where inflammatory symptoms are marked, 2 per cent solution of silver nitrate suits admirably, but it should be replaced by bluestone as soon as possible. Other escharotics are—corrosive sublimate solution (1 to 4 per cent), solid lapis divinus, and undiluted carbolic acid. *Operative measures* are to be adopted under the following circumstances:—(1) When large masses of granulations are present; (2) when the cornea is affected; (3) when local treatment has been tried for several months without success; (4) when a patient is unlikely to submit to a prolonged course of escharotic treatment; (5) when one eye is alone involved. The various operations include expression, partial removal of the upper retro-tarsal folds, and scrubbing the palpebral conjunctiva with a tooth-brush dipped in some antiseptic lotion. The chief *accessory measures* are the use of such lotions as previously mentioned, and the treatment of corneal ulcerations.

PHLYCTENULAR CONJUNCTIVITIS

This condition is common in weakly children of the poorer classes, and frequently develops after measles or other zymotic disease. In many cases it is dependent on "scrofula" or "tubercle." Bach believes that it is set up by pyogenic microbes, particularly the staphylococcus pyogenes aureus. He has succeeded by inoculation in producing the same affection, not only in rabbits, but also in human beings. Its chief importance is derived from the fact that it is often associated with a similar affection of the cornea. It is extremely prone to relapse, unless its cause can be made out and remedied. Knies has suggested that it should be termed "eczematous" when it co-exists with eczema in other parts of the body, but that when such is not the case the neutral term "phlyctenular" should be retained.

Symptoms.—One or more small pimples appear upon the sclero-corneal junction, and a triangular leash of vessels, having its apex at the affected spot, is generally present. The little nodule speedily loses its epithelium, becomes converted into a superficial ulcer, and then heals completely. It may, however, resorb without at any time ulcerating. When phlyctenulæ lie in the ocular conjunctiva, at some distance from the limbus, they are usually large and accompanied by muco-purulent secretion. In children there are several atypical forms of phlyctenular disease that need not be described more particularly.

Treatment.—Improve the general health by good food, fresh air, cleanliness, outdoor exercise, plenty of sleep, and (whenever possible) by residence at the seaside or in the country. Cod-liver oil, best given as an emulsion, is of special value, and tonics, as Parrish's syrup and the syrup of ferrous iodide, are useful. The use of these remedies should generally be preceded by a short course of mercury, best given as: Hydr. c. cret., gr. 1; pulv. rhei, gr. 1; sodii bicarb., grs. 2: one powder every night for a week. Locally, calomel[1] may be sprinkled over the injected parts once or twice a day, or the same remedy may be given for home use as an ointment containing 20 grains to the ounce. Yellow ointment (grs. 10 to the ounce) suits many cases, and may be used for months together where there is a marked tendency to relapse. Should photophobia be prominent, atropine or cocaine may be combined with the mercurial preparation.

Spring Catarrh.—This rare form of disease is important from its liability to be confused with trachoma. Its signs and symptoms, however, are quite characteristic. It is marked by firm, grayish elevations in the limbus, as well as by a pale condition of the palpebral conjunctiva, which is so closely beset by enlarged papillæ as almost to recall the appearance of a cobble pavement. The palpebral conjunctiva looks as though it were coated with a thin layer of milk (Vetsch). The patient complains of great irritability of the eyes of a distinctly seasonable type. The irritation begins in spring or early summer, and, after subsiding more or less completely during the winter, returns during the following spring. Spring catarrh usually affects both eyes, although not necessarily to the same degree. It is met with mainly in young persons, and may persist for many years. Nothing definite is known as to its pathology.

Treatment.—Lotions containing boric acid, sulphate of zinc, or antipyrine are useful. Ointments of salicylic acid or corrosive sublimate (gr. 1-2 to the ounce of lanoline-vaseline), or of ammoniated mercury (2 per cent), may be employed. In some cases douches of hot water appear to give temporary relief. If photophobia exist, the eyes should be protected with goggles. Surgical measures have been recommended, but are not in favour with most ophthalmic surgeons. Arsenic may be given internally. For my own part, I have seen more benefit from change of air than from any other means, local or general.

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[1] Preparations containing iodine must be avoided if calomel is being applied, because there is a danger of damaging the conjunctiva if the two come into combination under the form of mercuric iodide.

Exanthematous Conjunctivitis.—By this name we mean any inflammation of the conjunctiva associated with, or closely following an exanthem. Measles often gives rise to such a condition, which is characterised by its tendency to relapse and its rebelliousness to treatment. It takes the form, as a rule, of a catarrhal ophthalmia, with a coincident eruption of phlyctenulæ and blepharitis; photophobia is a common feature. In addition to this, many outbreaks of measles are ushered in by conjunctival inflammation. During an attack of small-pox, pustules may develop upon the conjunctiva or cornea, and in the latter position may entail serious results. I have occasionally observed small pustules upon the conjunctiva and intermarginal space during varicella. Apart from acute exanthemata, certain chronic inflammations of the skin may be complicated with conjunctivitis. In acne rosacea a small nodule may form in the limbus, and be accompanied by localised redness and by photophobia. After persisting for several days the inflammatory signs disappear, but a clear, bleb-like spot may remain indefinitely. The conjunctiva may be accidentally inoculated with the vaccine virus, although such a result is commoner upon the edge of the eyelids. In a case reported by Cargill, firm, pale, flat swellings in the ocular conjunctiva were found with a persistent form of urticaria; they were noticed to vary in size from day to day. Characteristic conjunctival changes are present in leprosy. Lastly, eczema of the skin or scalp often coexists with phlyctenular affections of the conjunctiva, altogether apart from the pus inoculations mentioned upon an earlier page.

Tuberculosis

Tubercle of the conjunctiva is met with clinically under several distinct forms. It is often primary—not associated, as far as one can tell, with tubercle in other parts of the body—and in that event is almost certainly the outcome of an external infection. This rare affection runs a lingering course, is usually limited to a single eye, and seldom attacks subjects over twenty years of age. It may or may not coexist with lupus of the face or elsewhere. The form most commonly recognised is characterised by the development in the conjunctiva of small, yellowish nodules, or of bleeding, cockscomb-like granulations, which sooner or later ulcerate. The nodules may be found in any part of the conjunctiva, but the flattened granulations are often confined to that of the lids. The resulting ulcers have ragged edges, gray, nodular bottoms, and are often more or less hidden by pus or debris. The eyelids become swollen, heavy, and unsightly; the conjunctiva throws off a muco-purulent secretion; pain or discomfort appears to be more often absent than present. In advanced cases the cornea may become involved. Erosion of the eyelids may occur, and adhesions between the ocular and the palpebral conjunctiva have been seen. The neighbouring lymphatic glands are practically always enlarged and tender. In another form the disease appears under the guise of a pedunculated tumour, attached by its pedicle to some portion of the palpebral conjunctiva. It resembles a papilloma more than anything else.

Diagnosis.—The diagnosis turns upon the discovery of the tubercle bacillus in the discharge or pieces of the diseased conjunctiva. In obscure cases inoculation experiments or the tuberculin test prove useful.

Treatment.—An attempt must be made to extirpate the whole of the granulations, as by excision, scraping, or the use of the galvano-cautery; after the operation, iodoform should be applied locally. Recurrences should be watched for, and when found, promptly attacked. *Internally*, creasote and cod-liver oil, separately or combined, appear to be of service. Careful attention must always be paid to the sanitary environment of the patient.

SYPHILIS

The initial lesion, or chancre, is now and again observed upon some part of the conjunctiva, especially in children. It forms a round or oval ulcer, the edges of which are usually raised; induration may or may not be present. The corresponding preauricular and angular glands speedily become involved. *Treatment.*—The affected mucous membrane should be washed, night and morning, with Lot. hydr. nigra (B.P.), and after that, the sore dusted with powdered iodoform. As soon as the diagnosis is made, general treatment by small doses of mercury should be begun. Several instances have lately been reported of a curious *infiltration of the ocular conjunctiva* coming on during the secondary stage of syphilis, rendering the conjunctiva swollen and semi-translucent. *Treatment.*—Mercury internally and black wash locally. *Mucous patches* have been observed by several writers. *Gummata* are occasionally found in the ocular conjunctiva, and ulcers may be produced by their disintegration.

Ophthalmia Nodosa.—This rare affection is due to the penetration into the eye of the hairs of certain kinds of caterpillars (*Bombyx pini* and *rubi*). It is characterised by many small, hard, grayish growths not only in the lower part of the conjunctiva and sclera, but also in the iris and other parts of the eye. Inflammatory symptoms may attain a high grade. A history may be got of a caterpillar having been thrown against the eye; or the patient, by the nature of his occupation, may have been liable to an injury of that kind. Diagnosis will be rendered certain by excising a nodule for microscopic examination, when it will be found to consist of round and of giant-cells, together with a hair. *Treatment.*—The nodules should be removed, and inflammatory symptoms combated by sedative and antiseptic lotions.

DEGENERATIONS

Xerosis Conjunctivæ.—Xerosis occurs under two forms—the epithelial and the parenchymatous. Epithelial xerosis is a superficial change of the ocular conjunctiva, limited parts of which become dry and lustreless, and are covered with a foam-like, whitish substance, containing the xerosis bacillus in large numbers. The changes are specially prone to affect the temporal side of the ocular conjunctiva, and often take a triangular or oval form. The spots appear slightly raised, and (when magnified) give one the impression of being made up of so many minute globules of mercury. They may be readily wiped away, as with wool, but are reproduced within 24 or 36 hours. The neighbouring conjunctiva may be permeated with dilated vessels, and be thrown into small folds concentric with the edge of the cornea when the eye is moved. This form of xerosis affects mainly thin, poorly-nourished children, who not infrequently suffer from otorrhœa. It makes its appearance in spring or summer, and may recur year after year at that period without entailing any serious mischief. It may or may not be associated with definite night-blindness. It has been shown by the writer to coexist with several other conditions: (1) a deficiency in hæmoglobin; (2) alterations in the visual fields for green and red; and (3) an exaggeration of the fundus reflexes visible with the ophthalmoscope in most young eyes. We assume that epithelial xerosis is the outcome, remotely of lowered nutrition, and immediately of dazzling by bright light. *Treatment.*— The most important remedy is iron, best given in the well-known form of Blaud's pill.

Parenchymatous Xerosis.—This much more serious malady commonly forms part of some general ailment, the exact nature of which is not well understood. In this country it is found only in young children, who show, besides frothy conjunctival patches, night-blindness and cloudy corneæ. The corneal changes usually go on to perforation, and the patients nearly always die. This malady is generally bilateral, and the usual signs of irritation, such as redness of the eye and photophobia, are often conspicuous by their absence; indeed, the lacrymal secretion may be wholly suppressed. As causes, hereditary syphilis, insufficient or badly-selected food, and prolonged diarrhœa or vomiting, have been mentioned. Among natives in India Herbert found mucous membranes other than the conjunctiva involved—for example, those of the mouth, nose, larynx, bronchi, intestine, and bladder. *Treatment.*—The eyes should be cleansed with sublimate (1:5000) and covered with pads steeped in the same solution. Physostigmine sulphate, gr. 1; cocaine hydrochloride, grs. 4; distilled water, 1 ounce, may be dropped into the eyes two or three times a day. However, more success is to be looked for from the employment of general than of local measures.

Pemphigus.—The conjunctiva is occasionally affected by pemphigus, which may or may not be associated with a similar disease of the skin or of other mucous membranes, especially those of the lips, nose, mouth, and fauces. From time to time bullæ make their appearance upon the conjunctiva, which becomes, at first, thick and red, and, later, undergoes cicatricial contraction. It is rare, however, to see the actual blebs, because they are so delicate as to rupture speedily. In this way the conjunctival sinuses are shortened or altogether obliterated, trichiasis or entropion is set up, and the lids become adherent to one another or to the globe of the eye. The corneal epithelium also suffers, becoming dull and dry, like the eye of a dead fish. To this advanced condition the name "essential phthisis" or "essential atrophy of the conjunctiva" has long been applied. The condition is essentially chronic. As a rule, the prognosis is bad, although a few cases seem to recover. According to modern researches, pemphigus is due to a diplococcus, different from any of the ordinary pyococci, and capable of cultivation upon various media outside the human body. The acute form of the disease attacks butchers with disproportionate frequency (Pernet). It seems likely that the conjunctival affection may arise in one of three ways: (1) as a primary disease resulting from ectogenous inoculation; (2) as an extension, through the lacrymal passages, from pemphigus of the mouth or nose; and (3) by conveyance of the specific microbe by the patient's finger from cutaneous bullæ. *Treatment.*—The treatment is purely palliative. Arsenic may be administered internally, and the conjunctiva kept clean and supple with ointments containing corrosive sublimate (gr. ½), iodoform (grs. 5), or boric acid (grs. 60). Boroglyceride has also been employed. Attempts to remedy the condition by transplantation of mucous membrane from other parts of the body have so far not yielded very encouraging results.

Lardaceous Degeneration.—This rare disease has been observed chiefly by Russian surgeons. It is marked by a progressive, non-inflammatory hypertrophy, commencing in the retro-tarsal folds or plica semilunaris, and slowly spreading to other parts of the conjunctiva. The affected mucous membrane is of pale colour, resembles in appearance bacon-fat, and does not bleed when incised. It is said to attack adults, who may or may not have suffered from trachoma, and to occur independently of degeneration elsewhere, as in the liver, spleen, or kidneys. *Treatment.*—Excision, partial or complete, is recommended by those who have had experience of the disease.

Conjunctivitis Petrificans.—Under this name Leber has described a peculiar affection of the conjunctiva, which becomes chronically inflamed and bestrewn with white, opaque dots. The chalky-white colour of the deposits contrasts with the redness of the mucous membrane in which they lie. They gradually get larger, so that the parts involved finally look almost as though they had been petrified. Leber found the deposits to consist of an organic combination of chalk. It is noteworthy that in Leber's case the disease led to partial symblepharon, and that the mucous membrane of the tongue showed epithelial changes.

VARIOUS CONDITIONS

Pterygium.—A condition of the ocular conjunctiva, a fold of which assumes a triangular form, and becomes firmly adherent to the superficial layers of the cornea. The true pterygium occurs only on the inner or outer side of the cornea in the region corresponding to the palpebral fissure. A false pterygium, however, may be met with in other positions. At first, when the pterygium is growing, it is thick and fleshy and marked by obvious vessels, like the wing of an insect; later, it becomes thin and pale and almost of cicatricial appearance. Its course is essentially chronic. During the progressive stage, it gives an unsightly look to the eye, may mechanically limit the ocular movements, and may encroach on the visual part of the cornea. The condition, which is rare in women, is met with chiefly in middle-aged men. It specially affects those who have lived abroad, and is rather common in sailors, probably because they are exposed to vicissitudes of weather. *Theories.*—Arlt assumes that noxious influence, such as dust, causes the epithelium of the conjunctiva and cornea to be shed, so that a little superadded swelling makes those two structures to adhere and a point or tag of conjunctiva to become fastened to the cornea. Fuchs believes that it originates from a pinguecula which has gradually made its way into the cornea, and in so doing drawn a fold of conjunctiva with it. A kind of spurious pterygium may now and then follow acute ophthalmia, burns, or scalds. *Treatment.*—If the condition be stationary, and have not encroached seriously on the cornea, it is best left alone, but otherwise an operation must be performed. The pterygium is carefully dissected away from the cornea, and, its apex being folded upon itself, is retained under the ocular conjunctiva by a suture. The conjunctival wound must be closed as neatly as possible by means of a continuous silk thread. The cornea, it must be remembered, always remains cloudy at the spot where the pterygium was attached.

Symblepharon.—This condition is marked by one or several adhesions between the ocular and the palpebral conjunctiva. It is caused by anything that produces an ulceration of the opposed surfaces, *e.g.* burns, the action of lime, certain forms of acute ophthalmia, tubercle, pemphigus, and operations on the conjunctiva. It affects the lower more frequently than the upper lid. Different names have been applied according to its position, as symblepharon posterius and anterius. The former indicates that the union has involved the conjunctival fornices; the latter that those structures have remained free. Symblepharon totale means that the eyelids are adherent to the eyeball. Symblepharon, when marked, may cause disfigurement, irritability of the eye, limitation of the natural movements, or (rarely) interference with sight. *Treatment.*—Many operations, some of a highly ingenious character, have been devised. The simplest plan is to separate the lid from the eyeball by dissection, and then to cover the raw places with morsels of mucous membrane taken from the mouth. Another way is to make liberating incisions in the neighbouring conjunctiva, so that the edges of the wound in the ocular conjunctiva may be brought together with sutures. The treatment of symblepharon posterius is not so satisfactory.

Pinguecula.—Pinguecula, like pterygium, is seldom seen save in elderly persons. It takes the form of a small, yellowish elevation of triangular shape, situated in the ocular conjunctiva, usually on each side of the cornea. The tumour consists of dense connective tissue (which has undergone a hyaline change) covered with thickened epithelium. *Treatment.*—Removal, if called for.

Effusions into the Conjunctiva.—So loose is the texture of the ocular conjunctiva that effusions of blood or serum readily occur into its substance. In extensive hæmorrhages almost the entire ocular conjunctiva may be involved, and blood may even pass for some little distance beneath the epithelial layer of the cornea. It is not uncommon after squint operations for the iris to appear changed in colour, owing to a transudation of extravasated blood into the cornea. The subconjunctival hæmorrhage becomes slowly absorbed. Its main causes are two in number: (1) Fragility of the blood-vessels; (2) injury, operation, strain, or inflammation. *Treatment.*—Compresses of lead lotion. *Chemosis*, or distension of the conjunctiva by blood serum, may accompany severe inflammations not only of the conjunctiva, but also of other parts of the eye or of its surroundings. It is, therefore, merely a symptom. In a marked case the cornea is overlapped by the swollen mucous membrane, which also protrudes from between the eyelids. Another kind of chemosis, thought to be of non-inflammatory origin, is sometimes observed in elderly persons. Some of the patients suffer from chronic Bright's disease, but, as a rule, the cause of the œdema is obscure. Holmes Spicer has recently described a form of chemosis, which he considers due to "obstruction of the lymph streams." The patients give a history of acute rheumatism, and the conjunctival affection is associated with lacunar tonsillitis, glandular enlargement, and febrile movement. Lastly, chemosis has been noted by Swan M. Burnet after the internal administration of quinine.

Staining of the Conjunctiva.—The conjunctiva may be stained by several agents, such as silver nitrate, protargol, largin, and iron sulphate. Lead acetate may lead to local ulceration and to deposition of the salt.

Lithiasis of the Conjunctiva.—In gouty subjects deposits of uric acid may sometimes be found in the palpebral conjunctiva. Should they cause irritation they may be removed. Small, yellowish concretions are quite frequently found in the palpebral conjunctiva of young persons. According to Fuchs, these are to be looked upon as tubular glands of new formation containing fungi (*pilzrasen*).

Conjunctivitis from Dazzling.—A form of painful ophthalmia may quickly follow exposure to snow, electric light, or the X-rays. It is to be treated by cold applications to the lids, and by dropping a 2 per cent solution of cocaine into the conjunctival sac at intervals. It speedily gets well.

Emphysema.—This implies a communication between the subconjunctival tissue and the nasal cavity or air-sinuses about the orbit. Traumatism is the usual cause. On palpation, characteristic crepitation can be elicited. *Treatment.*—Compressive bandage for a few hours.

TUMOURS OF THE CONJUNCTIVA

Besides the tumours already mentioned the following new growths have been found in the conjunctiva: cysts, parasitic and otherwise, myxoma, papilloma, fibroma, polypi, epithelioma, sarcoma, and carcinoma. The treatment is to remove the tumour with the least possible disturbance of the neighbouring parts. A simple puncture, however, may sometimes suffice, as when dealing with the so-called "simple cyst." In the case of malignant growths (which generally originate from the limbus) it may be advisable to cauterise the spot of origin.

Injuries of the Conjunctiva

The conjunctiva may be injured as the result of direct violence or the action of chemical agents. The degree of inflammation is often out of all proportion to the size of the irritant. Mechanical injuries, doubtless, quickly become complicated with pathogenic organisms, from which the conjunctival sac is seldom free. Moreover, microbes may have been introduced upon the surface of foreign bodies or in other ways. Injuries from lime deserve special mention. They are very formidable when caused by quicklime, which has a double action, namely, that of heat evolved by contact with the tears and that of chemical irritation. When due to slaked lime, which is more commonly the case, they are less disastrous. *Treatment.*—In severe cases it is necessary to place the patient under the influence of an anæsthetic, and, after removing every particle of foreign matter, to apply castor oil to the injured mucous membrane. Prognosis will always be grave, owing to such secondary results as implication of the cornea, hypopyon, entropion, and symblepharon. Uncomplicated wounds of the conjunctiva heal kindly enough if first made aseptic, and then drawn together by fine silk sutures. It is said that the staining left by grains of gunpowder may be got rid of by electrolysis.

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Constipation.

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Physiological Considerations.—While the frequency with which the excreta of the bowels are expelled varies at different ages, it may be accepted as practically normal that from later childhood onwards an evacuation at least once daily should occur. It is true that in the case of many, especially of the female sex, defæcation occurs much less frequently—once in three or four days, or even seldomer,—but while it has to be admitted that many of such cases present apparently few or none of the symptoms of fæcal retention, yet none the less such a condition of the excretory functions can hardly be looked on as normal.

The intestinal contents, which in the small bowel are in a very fluid condition, undergo a marked absorption during their passage through the large intestine. Hence it is only in the latter part of the colon that they assume the characteristic semi-solid appearance of a natural motion, and in the sigmoid flexure the fæces are stored until the act of defæcation takes place. The incitement to this is usually the passage into the rectum (as a result of peristalsis in the sigmoid flexure) of part of the contents of the latter. This is apparently frequently induced by the entrance into the empty stomach of food, especially when the sigmoid flexure is moderately distended with excreta. [An exaggeration of this normal mechanism is seen in what is known as lienteric diarrhœa, where the ingestion of a meal is at once followed by a desire for evacuation as the result of the entrance of a small quantity of fæces into the rectum from the merely partially loaded sigmoid.]

Although the act is a reflex one carried out through a centre in the spinal cord, it is largely influenced by the will. In man this centre is situated in the lumbar region of the cord. The presence of the accumulated fæces in the rectum causes, by reflex action through the centre, relaxation of the sphincter, while increased intestinal peristalsis is also set up. In voluntary defæcation the act is started by a full inspiration, closure of the glottis and fixation of the diaphragm, followed by contraction of the abdominal muscles and levator ani, the latter in this way exerting pressure on the rectum, while the tension of the pelvic fascia and muscles of the pelvic floor offer the required resistance.

Definition.—Constipation may be defined as a condition in which the alvine evacuation is difficult and deficient either in amount or frequency, or both, with a consequent tendency to fæcal retention.

While in reality largely a symptom present in a great variety of conditions and not a disease, constipation is frequently in itself of so much importance as almost to warrant its being regarded as such. Certainly in view of the great frequency of its occurrence, the amount of discomfort and suffering it produces, as well as the beneficial effects and satisfactory results of its proper treatment, the condition is one which calls for more careful attention and less empirical treatment than it often receives from the practitioner. (For constipation in children see "Gastro-Intestinal Disorders.")

CAUSES.—While the causes of constipation are so numerous and varied that it is difficult to summarise them, it is essential to the satisfactory treatment of any case that the real source of the trouble be discovered. As a rule several factors are at work, and these may perhaps be best considered briefly under two heads:—(A) Faulty expulsive mechanism; (B) Faults in the intestinal contents.

Faulty Expulsive Mechanism.—In this first group of causes one of the most important is that of *habit;* carelessness in attending to the call to stool, with the result that the bowel becomes accustomed to the presence of fæcal accumulations, and one of the chief stimuli to regular defæcation is thus lost. In other cases the sedentary habits or occupation of the patient, with the consequent feebleness of the abdominal muscles and sluggishness of the whole circulation, constitute a powerful factor.

In another group of cases the source is to be found in the *bowel.* Apart from the existence of actual constriction from malignant disease in the bowel, the possibility of which must never be lost sight of even in comparatively young subjects, constipation may arise from feebleness of peristaltic force consequent on a lowering of the nervo-muscular tone of the bowel, *e.g.* in old age or anæmia, from cicatrisation of old ulcers, from the presence of peritonitis (subacute or chronic), or old-standing bands or adhesions (impeding peristalsis), from pressure on the bowel by tumours or other swellings of adjacent parts, *e.g.* uterine enlargements, physiological or pathological, ovarian tumours, etc.; while again the over-distension which may result from such blocking of the lumen of the bowel still further tends to perpetuate the malady by lessening the peristaltic force. In other instances, especially of painful pelvic disorders in women, the dread of pain in defæcation and actual pain itself may have an inhibitive effect on peristalsis, and the same is seen in cases of hæmorrhoids, anal fissure, etc.

On the other hand, the constipation may arise from an interference with the nervous mechanism concerned in controlling the act of defæcation, from involvement of the centres or nerve-fibres, as in some forms of spinal disease, or as a part of various cerebral disorders, *e.g.* melancholia, tubercular meningitis, etc.

Lastly, *inefficiency of the accessory muscular mechanism* may contribute to the production of constipation, as in some cases of paralysis, or in patients whose abdominal muscles have been over-distended, *e.g.* by repeated pregnancies, ascites, etc.; or again, the presence of severe cardiac or pulmonary disease may render any straining efforts on the part of the patient impossible.

Faults in the Intestinal Contents.—The solid as well as the liquid ingesta may be at fault, or, on the other hand, the intestinal secretions may be defective. Thus the food taken may be too soft and too easily absorbed, leaving little or no residue, such a diet in many cases having been adopted because of deficient masticating powers owing to loss of teeth. Or the intestinal contents may be too dry, either from insufficiency of liquids taken or deficiency in the intestinal secretions. Instances of this latter are seen in cases of jaundice, diabetes, etc., or cases of excessive perspiration (from exercise or disease), bringing about increased loss of fluid from the body. In other instances the *quality* of the drinking-water is at fault; thus it may contain an excessive amount of lime salts, or it may be contaminated, *e.g.* with lead.

SYMPTOMS.—When constipation is merely one of the manifestations of the presence of some other malady (*e.g.* tubercular meningitis) its symptoms are scarcely to be separated from those attendant on the primary disease. But while it is undoubtedly true that in many instances of habitual constipation the patient exhibits little in the way of symptoms, yet in the majority of cases there is found to be present to a greater or less extent such manifestations as general languor, torpidity, and depression of spirits, along with irritability, a feeling of fulness in the head, passing into actual headache, furring of the tongue, with foulness of breath and loss of appetite, together with some feeling of fulness and distension in the abdomen, or even pain. In a few cases marked mental disturbance is present. Severer cases may be attended by vomiting, hiccough, etc., the usual symptoms of intestinal obstruction.

The characteristic stools in constipation consist of small, dry, hard, often dark, and offensive masses, along with which, in cases of old standing, is occasionally to be found mucus, or even blood, if severe straining has been required for their expulsion.

Lastly, it must never be forgotten that *diarrhœa* may be really a symptom of constipation, the accumulated fæcal masses by their presence giving rise to catarrh in the lower part of the bowel, with consequent desire for frequent evacuation. In such cases it is often only on digital examination of the rectum that the existence of constipation is revealed, as shown by the continued presence of scybalous masses in the rectum in spite of frequent evacuations. In addition, what may be termed secondary symptoms of constipation may arise, either locally, *e.g.* prolapse of the bowel, hæmorrhoids, etc.; or as the result of pressure on the surrounding parts consequent on the loaded condition of the bowel there may be vesical or uterine catarrh; or, again, varicosity of the veins of the leg (especially the left) may be increased, if not actually set up.

Before commencing treatment it is important, in view of the difference of prognosis in the two conditions, to decide if possible whether the case be one of mere functional derangement or of organic disease.

TREATMENT.—As has been already insisted on, this must be rational, and adapted to the particular causes at work in each case, and to this end the relatively subordinate position of *drugs* in the treatment of the majority of cases should always be borne in mind. The mere routine ordering of some cathartic remedy, without further attempting to prevent the recurrence of the constipation, cannot be too strongly condemned. The enormous and increasing consumption of innumerable patent medicines indicates only too clearly the extent of the evil which is unfortunately too often only perpetuated by their injudicious employment. The administration of a vigorous cathartic, however, as a commencement to the thorough treatment of a case of constipation is often not only very beneficial but even essential; but it is always advisable to explain fully to the patient the general hygienic and dietetic management of the condition before advising the employment of medicinal remedies.

Habit.—This is one of the most important prophylactic measures in the treatment of constipation. The patient must be urged to go to stool each day *regularly at the same hour*, preferably after a meal, but the time should be chosen with a view rather to its being available *every day* for this purpose. Thus for those whose business requires them to take breakfast at varying hours—often with a hurried rush for trains, etc.—the habit of going to stool at bedtime should be recommended; similarly those who suffer from severe anal troubles, and especially prolapse or piles, should be advised to choose the same hour. Not only will the patient be likely to obtain then greater facilities for sponging himself and returning the protruded mass, but the subsequent rest in bed allows of the congestion subsiding before active exertion is again called for.

It is of the utmost importance that this habit of punctual daily evacuation of the bowels should be insisted on from early childhood in both sexes. Too little attention is paid to this matter, especially in girls, entailing much subsequent annoyance, and even suffering. Those who have not acquired this regular habit must be encouraged to persevere in going regularly to stool at a fixed hour, even although no motion may result at first. In some cases the adoption of the more "crouching" attitude natural to defæcation in the open air—as by using a chamber-pot—has been recommended.

Again, with many, smoking after a meal acts decidedly as a laxative, but the all-important point is *regularity* and *punctuality* in the daily evacuation.

Diet.—The patient's diet should always be carefully inquired into. Foods made of the coarser grains, or in the preparation of which part at least of the more indigestible cellulose and fibre is retained, should be substituted for those of the finer sort. "Whole meal" porridge, oatcakes, and brown bread are usually easily taken, and the patient should be encouraged to eat the crust, and not merely the softer parts of the bread. Many people require syrup or treacle to their porridge, and these also are useful laxatives.

Vegetables, by reason of the large proportion of cellulose and neutral salts they contain, are also very helpful, while the gases which some of them are apt to evolve still further stimulate intestinal peristalsis, though they may somewhat increase the patient's flatulent distension. Thus raw tomatoes, salad, or merely fresh lettuce, should be recommended, and as a general rule the various vegetables in season should enter largely into the regular dietary.

Similarly fruit, raw or cooked, should be utilised freely. An orange or a raw apple before breakfast is an efficient laxative to many, and the patient should be advised to see that fruit always forms a part of his daily dietary. The attraction which soft (non-laxative) milky foods have for those whose teeth have failed them must be counteracted by combining with these such fruits as stewed figs or prunes, apricots, etc., and if the fibres of these prove too tough for the patient's imperfect teeth artificial ones must be obtained. Fruits in the form of jams also are of advantage, and marmalade possesses marked laxative properties. Milk and milky foods should be sparingly used, and of these latter the coarser-grained varieties, *e.g.* sago or whole rice, are preferable to such as arrowroot, and cornflour.

Liquids.—The importance of attending to the question of the amount of liquid consumed by the patient can hardly be overestimated. Many people, especially women, seldom take a drink of water, tea—too often with a fairly strong proportion of astringent tannin in it—being almost their only beverage. A single tumblerful of water taken on an empty stomach on rising—or, if required, at bedtime also—will frequently, in conjunction with the other general directions mentioned, have the desired effect. If the patient perspires much he should be recommended to drink water still more freely, especially *between* meals. Excessive "hardness" of the drinking water from the presence of lime-salts must be remedied by boiling the water and filtering, or by the use of rain-water for cooking purposes as well as drinking.

Coffee or cocoa are preferable to tea, containing less of the astringent tannin, and malt liquors tend to be more laxative than spirituous liquors. Buttermilk also may be ordered as being more laxative than ordinary milk.

Exercise.—The frequency of constipation in those of sedentary habits is well known, but the mere ordering of exercise is not enough. Care must be taken to see that the patient adopts some form of exercise likely to have a beneficial effect upon his abdominal muscles, and so on the underlying intestines. Thus riding on horseback, cycling, golfing, tennis, and swimming are much more likely to benefit than merely walking; while special gymnastic exercises calculated to strengthen the abdominal muscles (often so feeble in females) are of the utmost value.

One precaution, however, must be borne in mind. Should exercise, as in the case of some delicate women with pelvic disorders, give rise to pain, the constipation will probably be rather increased than diminished, the irritation inhibiting peristalsis apparently reflexly through the splanchnics. In such cases even moderate exercise may have to be prohibited, and (as afterwards referred to) an opiate may be required as a laxative.

Massage, etc.—This is frequently of most service in those cases in which the beneficial forms of exercise cannot be carried out, viz. the elderly or the very young. Especially useful is regular systematic massage of the colon in the direction of peristalsis, carried out by the patient himself or a trained attendant. Others have strongly recommended *vibration* as being a more potent and less dangerous mode of stimulating peristalsis in severe atonic cases. With this may be combined the use of cold compresses or cold douches to the abdomen, while in some obstinate cases beneficial results are obtained from the regular use of *electricity* to restore tone to the intestinal walls as well as the abdominal muscles. For this, special forms of electrical apparatus have been devised.

Medicinal Remedies. — Should the various hygienic and dietetic measures above referred to fail to effect a cure of the condition, recourse must be had to medicinal remedies, but only as an *adjuvant.*

The number of cathartic or laxative remedies is enormous; here only a few salient points will be referred to.

(*a*) *Enemas.*—Where the constipation is of long standing, with an accumulation of hard scybalous masses in the rectum and sigmoid flexure, these should be softened by enemas of soapy water or olive oil and oxgall, while in severer cases it is sometimes necessary to break down the masses and remove them by means of a spoon handle, care being taken to see that the accumulation is thus thoroughly removed. Should some scybalous masses be lodged higher up in the intestine, large douches of warm water inserted by means of a long tube connected with a head of water should be persistently employed. It is well to remember that some writers have pointed out that, once this form of treatment has been begun, there is a risk of auto-intoxication—apparently from the liberation of toxines from the softened scybala—if the douches are not administered *daily* so long as any masses remain in the bowel. Again, as a stimulant to defæcation small enemata of glycerine, or suppositories of the same, prove very useful, the forms of the latter now obtainable constituting one of the most convenient and least injurious therapeutic remedies we possess. Especially convenient are they for patients who are from home or travelling; but the habit of employing them systematically is to be guarded against. Occasionally considerable benefit may be derived from the use of *small* enemata of *cold* water, which appear to act by stimulating peristalsis.

(*b*) *Drugs.*—All cathartic remedies which have an *astringent* tendency as part of their after-effects should be rigidly avoided in treating habitual constipation, *e.g.* Gregory's powder. What should be aimed at in prescribing is to give something which may so tone up the bowel to act that the drug can be dispensed with in a short time. Especially important among these intestinal tonics are cascara sagrada, nux vomica, or strychnine, and aloes. These may be given together conveniently in pill form, a little belladonna being often added with or without hyoscyamus to prevent griping. It is most important to impress on the patient that it is not a *purgative* pill he is getting, but a *tonic* for his intestines, and therefore he is not to expect an action of the bowels as the result of one or two pills. Such a pill as—℞ Extr. cascaræ sagradæ, gr. iss.; extr. nucis vomicæ, gr. $\frac{1}{4}$; extr. aloes, gr. $\frac{1}{2}$; extr. belladonnæ, gr. $\frac{1}{6}$; extr. gent. co. q.s., given twice or thrice daily, *always as an adjuvant to the general measures stated previously*, seldom fails to bring about in a few days a regularity in the evacuations which continues after the pills have been *gradually* omitted. In anæmic females the addition of a little iron in some form to the pill is often of great advantage. Again, the liquid form of cascara (with which an equal amount of glycerine may be advantageously combined) is sometimes very effectual if taken nightly for some time to tone up the intestine, always in addition to dietetic and other general measures. The patient should be always strongly warned against indulging in occasional purgatives, and encouraged to persevere in measures calculated to bring about a natural daily evacuation.

Sometimes in plethoric subjects, or where the motions are excessively dry, salines given in the morning act best, either in the form of one of the numerous aperient waters, *e.g.* Hunyadi Janos, Rubinat, etc., or as the simple salts, *e.g.* Carlsbad, etc.; but while often beneficial for a time, and especially useful in obstinate cases as an alternative, *e.g.* during summer weather, they are never of the same lasting benefit as the purely intestinal neuro-muscular tonics, and if persevered in are apt to perpetuate the condition they were designed to cure. The advantage of sending cases of habitual constipation to one of the many purgative springs is doubtful. So far as the *cure* of his constipation is concerned the really important part of the treatment lies in his acquiring there those habits of regular hours, punctuality in attending to the call to stool, systematic exercise, etc., which he can follow out equally well at home. The action of the purgative waters is too often followed only by a recurrence of the constipation on their cessation.

Again, when there is sluggishness of the liver present, hepatic stimulants, *e.g.* euonymin, or podophyllin, are called for, but such drugs must be adopted as mere temporary additions to the systematic treatment of the constipation in the manner stated above.

Lastly, in some obstinate cases of constipation, as previously referred to, an opiate (often combined with belladonna) will open the bowels when various cathartics have failed, apparently by allaying the reflex inhibition of peristalsis; and in a similar fashion an opiate may be advantageously combined with a purgative if spasm or pain be a prominent feature in the case.

Contusions.

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Definition.—A contusion may be defined as a traumatic laceration of the subcutaneous soft tissues of a part, without solution in the continuity of the skin. When the integument gives way a *contused wound* results.

Causes.—The force producing a contusion is always applied by a *blunt* object, is momentary in its action, and immediate in its effects. It may be in the form of a direct blow acting perpendicularly to the surface, as in the case of a stroke with a stick, or a blow with the closed fist; or the force may impinge obliquely, as when a spent shot glances off the body, or a carriage wheel grazes a limb in passing. The damage resulting from a "percussing" force acting at right angles to a part is more or less localised to the seat of impact, but may extend deeply; while the effects of an obliquely directed stroke tend to be more diffuse and superficial. At the same time there may be much tearing of the subcutaneous tissues from the latter form of violence.

On the other hand, a part may be contused by "pressure," as when a limb is squeezed between buffers, or crushed by a waggon wheel passing over it. The resulting lesions are more widespread and deeper than in the case of injuries by percussion.

Morbid Anatomy.—Three degrees in severity of contusions are recognised.

In the *first degree* or *contusion with ecchymosis* there are small, interstitial, petechial hæmorrhages resulting from the rupture of minute blood-vessels in the skin, with slight œdema of the whole of the injured part. The effused blood is as a rule spontaneously and rapidly reabsorbed.

Second degree or *contusion with extravasation of blood.*—The extent and character of the effusion in contusions of the second degree depend upon the looseness or density of the connective tissue of the injured part, the size and nature of the damaged vessels, and the degree of force employed to produce it.

It may be that all the tissues are infiltrated with blood from a number of torn veins and small arteries; or that the blood from a ruptured artery or large vein collects in a single space, constituting a *hæmatoma.*

In either case the bleeding is soon arrested by the gradually increasing pressure in the effusion, and by the coagulation of blood in the torn vessels. The extravasated blood tends slowly to diffuse itself along the lines of least resistance—under fasciæ, between muscles, into vascular sheaths or connective tissue spaces—so that it may eventually reach the surface at some distance from the seat of injury. This fact is sometimes of importance in the diagnosis of injuries, especially in certain fractures of the base of the skull, where the ecchymosis appears after some days under the conjunctiva, or behind the mastoid process.

The majority of contusions tend to spontaneous cure. The fluid part of the extravasated blood is reabsorbed by the lymphatics. Some of the red corpuscles regain the general circulation directly or by the lymph stream. The solid clot disintegrates. Hæmatoidin crystals are precipitated in the tissues, giving them a bluish, green, or yellow colour; and crystals of cholesterin may form from destruction of the fatty elements of the clot. The remaining debris is carried off by the lymphatics, and may cause temporary enlargement and pigmentation of the adjacent lymphatic glands.

The destroyed tissue is replaced by a cicatrix formed by a process analogous to that known as healing by the first intention.

It is not uncommon for proliferation of the connective tissue surrounding an effusion of blood to take place to such an extent that an elementary membranous capsule is formed. In this way a *hæmorrhagic cyst* is developed, the contents of which may in course of time become absorbed, or, after drying up, undergo calcareous degeneration.

Certain contusions result in the outpouring of a large quantity of *serous fluid* instead of blood, most probably from tearing of large lymphatic vessels. This is commonest after obliquely directed blows or crushes, acting chiefly on the superficial structures, especially when a tense fascia underlies the skin. Such effusions are slowly reabsorbed, sometimes remaining unchanged for months.

In a few rare cases, especially where the contused tissue contains much fat, or where a fracture coexists, localised collections of an *oily fluid* form, and are slowly reabsorbed.

Third degree, or *contusions with complete disintegration of tissue.*—When the vulnerant force is extreme, and the resistance of the contused tissues slight, their structure may be completely destroyed. Muscular tissue, blood-vessels, nerves, and connective tissues are reduced to a pulp, the circulation through the part is suspended, and the vitality so depressed that necrosis takes place. The overlying skin becomes cold, livid, and dark-coloured, and blisters containing serous or bloody fluid may form on the surface. If septic infection be prevented dry gangrene takes place ; but if bacteria gain access suppuration and moist gangrene ensue.

Clinical Features.—The chief local phenomena of contusions are discoloration and swelling. When the condition is of the *first degree* minute punctate hæmorrhages are seen scattered through the superficial layers of the skin over the affected area. These are separate from one another, of a light colour, and with slight œdematous swelling around them.

Contusions of the *second degree* are those most commonly met with. The effused blood occurs in patches varying in size and depth with the degree of force which produced them, and in shape with the instrument employed. Where the skin is naturally thin or pigmented, as over the inner aspects of the arm, in the eyelids, scrotum, and perineum, the discoloration tends to be darker than elsewhere. When the extravasated blood is only separated from the oxygen of the air by a thin layer of epidermis or by mucous membrane, it retains its bright arterial colour. This is often well illustrated in cases of black-eye, where the blood effused under the conjunctiva is bright red, while that in the eyelids is almost black.

The discoloration may take some days to appear on the surface if the primary effusion has been deep-seated among the muscles and under strong fascial bands; and in these circumstances it may appear some distance from the seat of injury. The presence of blood extravasated deeply in the tissues can, however, often be detected by the firm, resistant, doughy swelling which exists. On deep palpation a peculiar sensation, closely simulating the crepitus of fractures, is sometimes transmitted to the fingers, and is liable to lead to errors in diagnosis.

The changes which take place in the effused blood lead to characteristic alterations in the colour of the contused part. In from 18 to 24 hours the margins of the blue area become of a violet hue, and as time goes on the discoloured area increases in size, and becomes successively green, yellow, and lemon coloured at its margins, the central part being the last to change its hue. The rate at which this play of colours proceeds varies so much, and depends on so many circumstances, that no time limit can be laid down. A large bruise over the arm of a healthy person may disappear completely in two or three weeks, while one of corresponding size and severity over the pelvis of a weakly patient will persist for as many months.

In all contusions of the second degree there is marked *swelling* of the whole area involved, especially when the subcutaneous areolar tissue is abundant and open, as in the eyelids, scrotum, penis, and labia.

In contusions of superficial parts there is always at first great *pain* and tenderness on pressure, but it soon passes off. When dense fasciæ, ligaments, or periosteum are involved these symptoms are more severe and lasting. *Traumatic neuralgia* along the course of a bruised nerve-trunk is not uncommon; and marked *hyperæsthesia* over a bruised area frequently persists for a long time. Although the degree of *shock* is not always proportionate to the severity of the injury, sudden *syncope* frequently results from severe bruises of the testicle, abdomen, or head, and occasionally marked *nervous depression* follows these injuries. The function of a bruised part is always seriously interfered with while the effusion and swelling last. The only evidence of general *constitutional disturbance* is a temporary elevation of temperature to 102° F., or even higher—a form of waste product fever. Among the rarer clinical features may be mentioned *general anæmia*, when the local effusion of blood is very great; *traumatic icterus*, when the blood pigment is deposited throughout the skin of the body; and *fat embolus*, which is usually associated with a complicating fracture.

The clinical features of contusions of the *third degree* are of secondary importance to those of the graver injuries with which they are usually associated—fractures, dislocations, laceration of large vessels, nerves, or muscles—conditions which in diagnosis and treatment overshadow the accompanying contusion.

TREATMENT.—The main indications are (1) to prevent the further effusion of blood; (2) to alleviate the pain; (3) to maintain the vitality of the damaged tissues; (4) to promote the absorption of the blood and lymph already extravasated; (5) to avoid, or (6) to combat, bacterial infection.

If seen immediately after the accident the part should be placed at absolute rest in a slightly elevated position. Firm elastic pressure through a thick pad of cotton-wool is of great value in arresting the effusion; and cold (in the form of ice), lead and opium lotion, or a weak carbolic compress, are useful adjuncts.

When extravasation has already taken place massage is the speediest and best method of dispersing the effused products. The part is deeply kneaded and rubbed in a centripetal direction once or twice daily, and the patient is encouraged to move the part freely after each sitting, in order that the muscular movements may augment the action of the rubbing. Any abrasion of the skin of course contra-indicates the employment of massage.

When the effusion is so large and so tense as to threaten the vitality of the tissues an incision may be indicated, but is only to be practised when the certainty of maintaining asepsis is assured.

Septic complications will be met on general principles by incision and drainage, or, if necessary, by amputation.

MEDICO-LEGAL ASPECTS OF CONTUSIONS.—Contusions have often very important medico-legal bearings. When called to see a suspicious case the practitioner should note (1) the date when the bruise is alleged to have been inflicted; (2) the date of the examination; (3) the degree of the contusion and the precise colours exhibited at the time of examination, as an indication of the approximate age of the ecchymosis; (4) the shape of the discoloured area, in association with the character of the instrument with which it is alleged to have been produced; (5) the presence or absence of evidence of such complications as fracture, dislocation, external wounds, or injuries to internal organs.

It is well to bear in mind that those suffering from scurvy, hæmophylia (bleeders), and fat, anæmic persons in a low state of health may have extensive ecchymoses produced on their bodies by very trivial injuries, and further that ecchymosis may occur, apart from external injury, from violent muscular efforts, as in attempting to recover one's balance, or during severe vomiting or other form of straining. It is also worthy of note that many old people with weak circulation have darkly discoloured patches on the legs and feet which closely resemble contusions, and might be mistaken for them on the cadaver. The necessity for distinguishing between bruising and post-mortem lividity need only be mentioned.

On the other hand the absence of ecchymosis does not prove that no external violence has been inflicted, as severe blows, especially over the abdomen and thorax, are often not followed by external signs of bruising, even although internal organs are seriously damaged.

Violence inflicted on a living body may not be manifested by ecchymosis till after death, while blows dealt on a recently dead body (within two hours of death) may produce signs exactly like those occurring on the living. If, however, the body be cold and cadaveric rigidity has set in, the appearances of contusions on the living cannot be produced by external violence.

If a bruise on a dead body be found uniformly blue or livid, the presumption is that the blow was struck immediately before death, while a play of colours round the margin suggests that some time has elapsed between the infliction of the injury and death. That the effused blood has remained fluid is not proof that the contusion was sustained after death.

Evidence of the previous existence of a bruise may be found for some time in the pigmentation of the neighbouring lymphatic glands.

Convulsions, Infantile.

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DEFINITION.—The term convulsions is applied to more or less general purposeless muscular contractions, occurring simultaneously and successively for a variable time, attended in most instances by a loss of consciousness more or less profound. The more strictly local muscular contractions are more appropriately and conveniently denoted as spasm.

FREQUENCY.—The frequency of convulsions in infancy has been greatly exaggerated, especially amongst the lay public.

The convulsive movements of the limbs seen in moribund children, mainly due to the venous state of the blood, must not be considered in the same category.

ETIOLOGY.—The causes of convulsions can be divided into predisposing and exciting, of which the former are the more important.

Predisposing Causes.—1. All writers seem inclined to place as foremost amongst the predisposing causes what has been termed the *instability* or *irritability* of the infantile nervous system. This instability disappears with advancing age and the further development of the higher centres.

2. An inherited neurotic tendency or constitution is a very important factor. Published statistics give a neurotic history in 32 per cent of the parents; and if convulsions in brothers and sisters of the patients are considered, then the family neurotic history rises as high as 67 per cent. The importance of this factor in the causation of convulsions has not as yet been sufficiently recognised and acknowledged.

3. Rickets is a further predisposing cause. Whilst, however, a very large percentage of convulsive infants are rachitic, yet convulsions do not occur in certainly more than 5 per cent of all cases of rickets. When convulsions occur in this last they are frequently associated with laryngismus stridulus and tetany. The explanation of the occurrence of all three disorders is, perhaps, due to the brain suffering in the general mal-nutrition with the rest of the body in rickets.

Exciting Causes.—Almost any central or peripheral lesion in infancy may act as an exciting cause, especially in subjects predisposed. The more common conditions may be thus classified:—

1. Local disease or injury of the brain or its membranes, *e.g.* intracranial tumour, hæmorrhage, meningitis, etc. The symptoms of cerebral tumour in young children often present themselves with a startling abruptness, and convulsions at the onset may be general in character. If the convulsions date from birth they are generally due to meningeal hæmorrhage, and are often associated with paralysis and subsequent amentia. In not a few cases of meningitis, convulsions may be the first prominent symptom, and such convulsions may be unilateral in character.

2. Altered vascular states, as anæmia, following hæmorrhage or exhausting disease from any cause, venous engorgement, or, more rarely, uræmia.

3. The onset of the acute infectious diseases, *e.g.* pneumonia, etc. The frequency of this relationship has been largely overestimated, convulsions being much more frequent *in the course* of such affections, and then usually dependent on the venous state of the blood (asphyxia).

4. The developmental conditions resulting in idiocy.

5. Peripheral nerve irritation. Teething disorders and gastro-intestinal derangements are important factors in predisposed subjects. Severe surgical injuries, such as extensive burns, may certainly cause convulsions in infants in whom no predisposition is to be found.

6. Asphyxia. The important part played by asphyxia is perhaps not duly appreciated by the profession generally. This association clinically between asphyxia and convulsions is in strict accord with physiological experiments. Illustrative examples have already been given. The convulsive movements seen in various moribund states are probably also of similar origin.

PATHOLOGY.—The real pathology of convulsions is as yet undetermined. The post-mortem changes found in the brain and cord are in all probability results and not causes of the disorder. Probably the ultimate pathological processes concerned will be found in molecular changes in the nerve cells, rather than in changes capable of demonstration with any means yet at our disposal. When the pathology of idiopathic epilepsy is revealed to us, the discovery of that of infantile convulsions will be close at hand.

SYMPTOMS.—The phenomena of a typical attack of convulsions are identical in character, time, and sequence with those occurring in an epileptic fit, and require no detailed description. In both disorders the attacks generally commence with a dazed or "far away" expression of the face, immediately followed by momentary pallor and dilatation of the pupils. These are closely followed by loss of consciousness, during which the muscles are affected at first with tonic spasms and apnœa, succeeded after a variable interval by clonic contractions. In both convulsions and epilepsy there is frothing at the mouth, blood-stained if the tongue be bitten, and in both there may be involuntary discharge of urine and fæces. The attacks always conclude with a period of drowsiness more or less profound. Several of the phenomena may be but slightly prominent or even omitted, and convulsions, like epilepsy, may vary in their intensity between the widest extremes. A conspicuous feature of infantile convulsive attacks is the well-known turning in of the thumbs upon the palms of the hands, with flexion of the fingers around the thumbs. Convulsions may come on suddenly in the midst of apparently perfect health. In other cases there may be warnings in the shape of fretfulness, squinting, grinding of the teeth, and turnings in of the thumbs upon the palms of the hands.

DIAGNOSIS.—If the child be seen in the attack the diagnosis can present no possible difficulty. When it has to be made from the verbal accounts of the parents or friends, then the utmost caution and discrimination will have to be used. The too ready acceptation, without careful inquiry, of the laity's interpretation of what constitutes convulsions, has helped to spread and perpetuate an exaggerated impression of the frequency of convulsions proper even amongst the members of the medical profession.

Prognosis.—The immediate prognosis is generally favourable. When the attacks so rapidly follow one another as to be merged into one long series (status convulsivus), then death may ensue from exhaustion. This last condition—"status convulsivus"—differs in no respect from the "status epilepticus" of adults, except that in the younger subjects the temperature runs much higher and generally reaches 105° or upwards. Death, too, is not very infrequent in convulsions associated with laryngismus stridulus, but here the last-named is possibly more often responsible for the fatal issue than the concomitant convulsions. Convulsions arising during whooping-cough are often fatal, more especially if the infant's health has been much reduced by vomiting or other complication. When bronchopneumonia is marked by urgent dyspnœa, convulsions, due to asphyxia, are not infrequent and are generally fatal, but the conditions preceding them in this instance are of extreme gravity independently of them.

In most cases *the remote prognosis* of an attack of convulsions should be a guarded one. There is seldom anything in the phenomena of the attacks that furnishes any criteria enabling one to discriminate between convulsions likely to be followed by later neurotic manifestations, and those that are of merely temporary import. Prognosis then must depend less on the phenomena of the attacks than *on their history and the circumstances* which attend them. Here, however, it may be said at once that the same greater significance attaches to unilateral convulsive attacks in all cases in infancy as to those occurring in later life. Favourable factors are a history of previous good health in the infant, and the presence of some well-defined exciting cause, such as the onset of one of the exanthemata or croupous pneumonia. The prognosis is favourable, too, in cases where the convulsions can be shown to have mainly depended upon asphyxia, the cause of which has been removed or recovered from. Where there is a family neurotic history the prognosis should be guarded in the extreme. Caution should be used, too, in pronouncing as to the future of an infant who has suffered from convulsions presumably from such slight causes as dentition, bowel disturbance, or ascarides, for it may well be asked whether a nervous system that has once broken down under such slight causes would not be likely to do so again in the future at any times of cerebral stress or strain beyond the ordinary. Convulsions occurring in the course, not at the onset of scarlet fever, and which are independent of any kidney involvement, are often unilateral at the commencement, and are frequently continued into later life as epilepsy. When the convulsions occur in association with rickets, improvement in the last complaint generally leads to the final disappearance of any disturbance of the nervous system. But even here Sir William Gowers has shown that 10 per cent of adult epileptics have their start in infantile convulsions due to rickets, and in juvenile epileptics the proportion is probably greater. This should lead to a more guarded prognosis than is usually given in cases of rachitic convulsions. Cases where convulsions recur from time to time without organic cause are to be regarded in the most serious light, a large proportion of them merging into epilepsy in the near future, whilst others are probably the subjects of other neuroses in later life.

TREATMENT.—*During the Attack.*—In the majority of instances it is only necessary to loosen the clothing about the neck, chest, and abdomen, and to lay the infant in the supine position with the head slightly raised. The infant can then be left to recover from the drowsiness naturally following, or forming part of, the attack. The ordinary domestic remedy, placing the child in hot bath, does no harm probably if the child's health be not too enfeebled. In this last condition, instead of a plain hot water bath, a mustard one might be tried with advantage for the sake of its well-known stimulating properties. There is nothing to be said in favour of the other routine remedy, the application of cold to the head, except in the cases where it is used for the express purpose of lowering the temperature.

If the unconsciousness be unduly profound, and especially if it be attended with other fits, or threatenings of them, then recourse must be had to more active measures. First amongst these is the *inhalation of chloroform,* which is warmly advocated by Henoch, John Thomson, and other authorities. Profound unconsciousness is no bar to the use of the drug, and usually recovery from the anæsthetic is accompanied by the regain of consciousness. Eustace Smith praises the hypodermic injection of morphia in these cases in doses of $\frac{1}{24}$ of a grain to an infant of six months old, and says it can be safely repeated, if necessary, in the course of half an hour. The inhalation of *nitrite of amyl* in one minim doses has been recommended by some authorities. The administration of *chloral* by enemata, in doses of about three grains to an infant of six months old, is a valuable remedy for two reasons. If the drug be retained its sedative action on the nervous system is usually quickly manifested. In other cases its presence in the rectum leads to the evacuation of an especially foul-smelling motion, and this is generally followed by a speedy return of consciousness. If there be any valid reason for suspecting indigestible food to be responsible for the disturbance, then an emetic may be given, followed by a calomel purge. In very exceptional instances there may even be some justification for the employment of the gum lancet.

Subsequent Treatment.—For a few days after a fit small doses of one of the bromides, two or three grains, should be given thrice daily. If the child be syphilitic or rickety then the appropriate treatment for these complaints should be vigorously employed. The greatest care should be bestowed on the diet, and on the thorough ventilation of the day and night apartments. Arrangements should be made for the child's head being raised during sleep and the feet being kept warm. If the convulsions recur with any frequency, say, every fortnight, or even every month, then a course of bromides should be prescribed. Every attack possibly lessens the resistance in the nerve tracks, so that succeeding fits occur with much slighter exciting causes than the initial ones. By allowing the fits to continue much more harm may thus be done than any that may possibly arise from the effects of the bromides on the developing tissues. It is difficult to fix a definite time, but perhaps a freedom of three months from fits should ensue before stopping the bromides. Belladonna, the salts of zinc, digitalis, ergot, musk, and other remedies that have been vaunted from time to time in the treatment of convulsions, are much less efficacious than the bromides. If the bromides fail in checking the convulsions, then borax, in two-grain doses, might be given a trial.

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Coracoid Process. *See* Shoulder Joint.

Cornea.

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The cornea may be considered broadly as consisting of three layers: the *anterior epithelium*, the *proper corneal tissue*, limited by two structureless elastic laminæ, Bowman's membrane in front, and Descemet's membrane behind, and the *endothelium*. These parts have a common origin with the conjunctiva, with the sclerotic, and with the uveal structures of the eye respectively, and they are subject to the same changes and show the same tendencies as the parts with which they have origin. The cornea is not vascular; it gets nourishment by a system of lymph channels, and is abundantly supplied with nerves.

Examination of the Cornea.—The cornea should be generally inspected as to shape, size, clearness, and the presence or absence of opacities. Its surface should then be examined by placing the patient opposite a window, and observing the image reflected from the various parts of the cornea as the eye follows the finger. A breach of surface can be detected by a disturbance of the image; it may then be verified by staining with fluorescine. Fluorescine 2 per cent, bicarbonate of soda 3 per cent in distilled water. The condition of the cornea generally can be best made out by examination by focal light; finally, the ophthalmoscope may be used with a lens of high power + 20 D behind the mirror.

Examination by *focal* light is carried out as follows:—A darkened room, a source of light, and a lens of about three inches focal length are necessary. The light is placed at about eighteen inches on one side of the patient's head, and is focussed by the lens on the different parts of the cornea; the more obliquely the light falls on the eye the more clearly will opacities be seen. To obtain full advantage of this method the cornea should be magnified by a lens of high power held in the other hand.

Inflammation of the Cornea.—*Keratitis.*—Inflammation of the cornea may be limited to the epithelial layer, to the substance of the cornea, or to the endothelium on its posterior surface; it may be a local infective process leading to ulceration or abscess, or it may be part of a disease affecting mainly the deeper regions of the eye; it is often the manifestation of a constitutional affection as in inherited syphilis. Inflammation of the cornea always gives rise to a loss of transparency; if only the anterior epithelium be involved, the surface loses its polish, and the appearance of stippling produced by its cells when highly magnified is exaggerated; sometimes the change in the cells is so marked that the magnified surface has the appearance of a piece of orange skin or shagreen. An exudation into the corneal tissue proper is shown by a yellow or gray opacity; suppuration is preceded by haziness, and the area of suppuration is surrounded by a similar area of haziness. The effect of inflammation is to produce a blurring of the parts seen through the cornea; thus the pupil is no longer quite black, and the details of the structure of the iris are obscured.

Inflammation begins with an infiltration; this consists in the appearance of numbers of leucocytes in the tissue of the cornea; the affected area is rendered opaque and the surface over it loses its polish. This exudation sometimes becomes completely absorbed, and the cornea is left quite transparent; at other times where the disturbance of the corneal tissue has been greater, or where the exudation has been present for a long time, complete restoration of transparency does not take place, the exudation is converted into fibrous tissue, which differs from the proper tissue of the cornea in not being transparent. There is a tendency, however, for old exudations to go on slowly clearing for years, as is seen in the remarkable way in which the changes in the corneal substance left by interstitial keratitis disappear. This clearance depends probably on nutrition, as we commonly see the periphery of the cornea clearing more readily and more completely than the central parts which are farthest removed from the sources of nourishment. This also probably explains the reason underlying the use of irritating remedies and massage of the cornea in the promotion of clearing of opacities of old standing.

ULCERATIVE INFLAMMATION.—If the inflammation be of an ulcerative nature the exudation goes on increasing in amount, the corneal tissue is broken down, and either an ulcer or an abscess is produced. Suppuration generally starts in the superficial layers of the cornea; the epithelium becomes cast off from the infiltrated area and an ulcer is formed; in the earlier stages, while the margins or base of the cornea are infiltrated with leucocytes, the ulcer is *progressive.* As recovery takes place the area of infiltration disappears, the ulcer becomes clear and transparent, but the surface is still rough. Gradually the epithelium from the edges grows over the surface of the ulcer, and blood-vessels run in from the margin of the cornea beneath the epithelium; the ulcer is healed, but its level may be lower than that of the rest of the cornea. By the development of fibrous tissue beneath the newly-formed epithelium the level is gradually raised, but the transparency of the cornea is impaired by this growth of fibrous tissue, and the seat of a former ulcer may be traced for years by the opaque scar lying in the clear cornea.

Most of the pus-producing organisms have been found in ulcerative affections of the cornea. In the early days of bacteriological work on the cornea the staphylococcus and streptococcus were thought to be the most frequent provocative agents, whether the ulceration was primary or the result of injury, but owing to the work of Uhthoff, Axenfeld, Morax, and others, we now know that they are present much less frequently than other organisms. The ulcerations which occur during the course of attacks of conjunctivitis are not due to secondary infection by staphylo or strepto-cocci, but are caused by inoculation of the cornea with the organisms of the conjunctivitis, and we may therefore find the gonococcus, the Weeks' bacillus, the diplobacillus, and others. The characters of the ulcerative process vary with the nature of the infecting organism, and although we are not yet in the position of being able to make a new classification of corneal ulcers on this basis, yet certain facts are well ascertained. According to the researches of Uhthoff and Axenfeld, in as many as 98 per cent of the cases of acute serpiginous ulcer pneumococci were present. In certain serpiginous ulcers running a much less acute course, attended by little or no pain, a diplobacillus resembling somewhat the diplobacillus of chronic conjunctivitis, but having some distinguishing characters, has been found (Petit). In bilateral infantile ulceration of the cornea (kerato-malacia) the pneumococcus has been found and also the streptococcus. The Aspergillus fumigatus has been found growing on the cornea in some rare instances.

The *histology of corneal suppuration* has been very thoroughly investigated by Leber, who by his observations on the cornea has added largely to our knowledge of the inflammatory process in general.

According to his observations the entrance of any pyogenic organism into the tissue of the cornea sets up hyperæmia of the parts round the cornea. This is brought about by the irritative action of the toxines developed in the corneal tissue by the growth of the organism. Very soon an invasion of white cells spreads in from the periphery of the cornea, each cell making its way through the lymph channels till it reaches the infected area. Here two things may happen: the cells either come to a standstill, killed by the intensity of the poison, and form a ring of pus surrounding the focus of infection; or where the poison is less intense, the whole of the infected region is invaded by the white-celled exudation.

The irritative action of the toxines spreads beyond the cornea to the iris and ciliary body; these become hyperæmic; the blood-vessels of the iris enlarge, the iris tissue becomes swollen, its fibres blurred, posterior synechiæ may form, and from the surface of the iris and angle of the anterior chamber an exudation is poured out which sinks to the bottom of the chamber and forms a hypopyon. This hypopyon consists of a fibrinous exudation with leucocytes, and is generally fluid, so that its upper limit is horizontal, and its position varies with that of the head. A hypopyon is sterile except when the anterior chamber has been opened, when it may become infected from without. The opinion was formerly held that the hypopyon was derived directly from the seat of ulceration in the cornea by the passage of the products of inflammation through Descemet's membrane into the anterior chamber; the active resistance of this membrane and of its endothelium render this manner of access to the anterior chamber impossible. The hypopyon may sometimes be seen extending up to the region of the floor of the ulcer as a yellow cord communicating at its lower end with the general mass of the hypopyon. At the first view this would appear proof of a communication between the base of the ulcer and the chamber, but examination of the process microscopically shows that the corneal layers near the ulcer become swollen, the endothelium shares in this swelling, some of its cells exfoliate, and a deposit of coagulated lymph and cells forms on the denuded surface, which by increase comes to communicate with the general body of the hypopyon. This condition is sometimes described as posterior abscess of the cornea; it very frequently ends in a perforation of Descemet's membrane, but in their earlier stages the cells of the posterior abscess are sterile and frequently show their origin from the uvea in containing pigment.

Results of Ulcerative Keratitis.—If the seat of the ulcer be in the substance of the cornea itself, and the proper corneal tissue be destroyed, a permanent opacity results, the new scar tissue cannot acquire the peculiar transparency of proper corneal tissue, although the scars and blemishes left by ulcers in early life may to a great extent disappear. Such opacities are called *nebulæ*, *maculæ*, or *leucomata* according to their density.

If the loss of corneal substance is considerable the intraocular pressure may cause the floor of the ulcer to bulge. At first Descemet's membrane being very elastic and resistant does not give way, but forms a hernia at the seat of ulceration; the appearance of such a hernia is that of a transparent bead on the cornea. The protrusion may flatten and become consolidated, or it may perforate; perforation is usually accompanied by a reduction of pain and irritation in the eye, but its results may be serious. The iris may come into contact with the back of the ulcer, or it may be prolapsed through the perforation. If the ulcer then heal, an adhesion of the iris to the back of the cornea will be left varying in thickness from a few filaments to a broad band of iris substance, *anterior synechia.* If after perforation the lens come into contact with the back of the inflamed cornea, an opacity is formed at the anterior pole of the lens, and an anterior polar or pyramidal cataract results. The cornea sometimes heals with much loss of substance; being then too weak to resist the pressure of fluids within the eye it bulges irregularly and forms an anterior staphyloma; this also occurs after total destruction of the cornea, the place of the cornea being taken by a thin layer of fibrous tissue united with the tissue of the iris. This weakened membrane may burst and collapse, and become permanently flattened and consolidated, or it may yield again, and again burst. At the time of its distension it protrudes between the lids like a button, and is a source of much distress; after collapsing it again ceases to give trouble for a time. After a large perforation it is not uncommon for the lens to escape, for the vitreous to become prolapsed, and for the globe to suppurate or to undergo slow shrinking.

Causes.—Infection of the cornea by micro-organisms is necessary for the production of the ulcerative process; in most cases this is preceded by a loss of surface epithelium. This may be the result of an injury such as an abrasion of the surface or the entrance of a foreign body; thus it is common among men exposed to injury from their trade, but it also occurs as the result of a wound, as by a baby's finger-nail; some of the worst cases are among agricultural labourers, those engaged in hedge trimming, or especially among those at work in the harvest-field. In a considerable number of cases mucocele or chronic inflammation of the lacrimal sac is present, and this with its decomposing contents offers a constant danger of infection to an unsound cornea. Ulcerative keratitis may also occur from infection of the small ulcers during an attack of phlyctenular disease, or in a herpetic eruption on the cornea. The cornea may become infected during gonorrhœal conjunctivitis, when the whole of the epithelium is sodden and diminished in vitality, also where the nutrition of the cornea as a whole, and especially of its epithelium, has been profoundly altered, as in keratomalacia. Finally, ulceration of the cornea may take place as the result of a blood infection, as is sometimes seen during an attack of small-pox, when pustules not infrequently develop on the cornea itself.

Paralysis of the facial nerve, leading to weakening of the orbicularis muscle and to insufficient protection to the cornea; corneal anæsthesia depending on fifth-nerve paralysis; the insensibility of the cornea in absolute glaucoma, and in the late stage of exhausting diseases such as cholera, may lead to a loss of surface from exposure, or from unconsciousness of the presence of a foreign body.

Treatment.—Before beginning treatment the cause of the ulcerative process should as far as possible be sought for. Foreign bodies should be removed from the cornea and the conjunctival sac, misdirected eyelashes should be pulled out, and the condition of the lacrimal puncta should be noted. If the corneal affection is secondary to disease of the conjunctiva treatment of the latter should be actively pursued; in gonorrhœal conjunctivitis the nitrate of silver treatment should be carried out as long as the condition of the discharge renders it necessary, care being taken to prevent injury to the cornea by the applications or by the necessary eversions of the lids. A mucocele or lacrimal abscess should be sought for, as this probably more than any cause tends to keep up activity in an ulcer; the lacrimal sac should be opened, and its contents washed out daily; the passage into the nose should be made free, and if necessary a style should be inserted in the nasal duct.

In most cases of ulcer of the cornea great benefit will be derived from the firm application of a compress to the eye; it greatly prevents movement of the lid and the constant winking and spasm which are so painful, and it ensures warmth and protection; the eye should be first covered with a pad of cotton wool and the bandage should be applied over it. Where there is much conjunctival secretion, as in the cases of abscesses secondary to purulent conjunctivitis, it is best not to bandage the eye, because of the danger to the cornea of keeping the infectious discharge pent up between the lids.

Heat applied to the eye is a valuable means of treatment, especially when the general level of nutrition is low. The most convenient method of applying heat is by hot bathing; simple hot water, or better, hot saline, containing 1 per cent of chloride of sodium, or a solution of 2 per cent of boric acid, should be applied to the eyes by pads dipped in the solutions kept as hot as they can be borne; this bathing should be kept up for a half an hour at a time, and should be frequently repeated. A good way of keeping the lotion hot is to insert the basin in which it is contained in a basin of very hot water which can be frequently renewed. A means of applying heat that is very valuable is a Japanese hand-warmer, a small oblong tin box containing a slowly burning cartridge; if this be applied to the eye by a bandage over a layer of cotton wool, the heat can be kept up without difficulty for any length of time. Caution must be mentioned here against the use of any lotion containing lead where there is loss of the corneal epithelium; an insoluble, densely opaque film of lead salts may be deposited on the ulcerated surface.

Atropine should be used so that the pupil may be kept fully dilated; iritis is present in many cases of corneal ulceration, and atropine is necessary, to prevent adhesions and to subdue the iritis; apart from this, however, atropine secures physiological rest of the eye by dilating the pupil and paralysing the ciliary muscle. The best means of using atropine is in the form of an ointment of the strength of four grains of the alkaloid to the ounce of vaseline; a small piece should be inserted inside the lower lid twice a day, or more often if iritis is present. The ointment is preferable to drops, as it remains longer in the conjunctival sac, and acts more thoroughly on the eye, with less risk of the unpleasant symptoms of atropine poisoning from its absorption into the system. Atropine should be used just sufficiently to maintain full dilatation of the pupil, it should not be smeared freely over the lids; sometimes where its use has been too profuse or too long continued it gives rise to a highly congested state of the skin and conjunctiva, a condition known as atropine irritation. Atropine irritation occurs generally in those who are peculiarly susceptible to its action, in such people the smallest amount gives rise to symptoms. Where much redness of the skin and conjunctival irritation comes on during the use of atropine this form of irritation should be borne in mind, otherwise it may be thought that the ulcer is worse and needs more atropine. The drug should be discontinued and some other mydriatic, such as scopolamine or duboisine should be substituted for it if necessary, but it is better to discontinue all mydriatics and use a simple ointment of boric acid for the irritated skin. It is sometimes useful to combine iodoform with the atropine in infective ulcers. Atropine 4 grs., iodoform ʒj, vaseline ℥j.

Eserine has been strongly recommended by some surgeons as a routine treatment for ulcers of the cornea. Where there is a possibility of perforation it has undoubtedly a valuable place, but speaking generally, its use tends considerably to the increase of irritative symptoms, pain, and intolerance of light, and to the circumcorneal congestion and iritis. It should be reserved for those cases in which imminent danger of a perforation of the cornea leads to the fear that an extensive prolapse of the iris may occur. Eserine is also of value in certain cases in which the ulcer is indolent for a long time, neither advancing nor healing—a condition depending, probably, on impairment of nutrition. The increase of vascularity in the neighbouring parts induced by eserine promotes the nutrition of the cornea, the ingrowth of blood-vessels, and the healing process in the ulcer. It may be used as an ointment, or as drops in the strength of one or two grains to the ounce.

Where the ulcer is progressive, with an advancing infiltrated edge, these measures are insufficient; recourse must be had to the *actual cautery* or to *strong caustic applications* to the seat of the ulcer. The cautery is most conveniently applied by a flattened loop of platinum wire bent to a convenient angle, and raised to a dull red heat by a galvanic current. The ulcer being stained with fluorescine all the parts which take on the stain should be burnt, especially where the infiltration is densest; it is not necessary to burn the whole surface of the ulcer, the part that is healing and covered with endothelium will not stain, and should not be touched.

If the galvanic cautery be not available, a small Paquelin's or a metal cautery heated in a spirit-lamp may be used, but these are much less handy. If the temperature of the wire be not greater than a dull red heat the cautery does not produce effects beyond the parts actually touched by it; if the thin base of the ulcer be perforated the incandescent wire is at once chilled by the escaping aqueous and no harm results.

For most advancing ulcers treatment by strong local applications is enough; the ulcer should be stained, the infiltrated parts should be scraped with a small sharp spoon, and a camel-hair brush dipped in the fluid should be painted over the stained surface, and introduced into all the pockets and recesses of the ulcer. Of these caustic applications pure carbolic acid is one of the most efficient and least painful; where it touches the cornea the tissue is at once whitened and afterwards cast off, so that it should not be applied carelessly or in excess. Other agents, such as nitrate of silver, 10 grs. to the ounce, perchloride of mercury 1 or 2 per cent, or tincture of iodine, may be used, but the pain from them as a rule lasts longer and is more severe than after carbolic acid.

Before using any of these methods of treatment the eye should be thoroughly cocainised and a speculum introduced; as immobility of the eye is very essential, a small amount of solid cocain in fine powder may be applied to the ulcer and also to the conjunctiva where it is gripped by the fixation forceps. After cauterisation atropine ointment or atropine ointment with iodoform should be used, and a compress. Section of the base of the ulcer, opening the anterior chamber, a method originated by Saemisch, is a very valuable treatment where a hypopyon is present, or when there is risk of perforation. A Graefe's knife is passed into the anterior chamber at one side of the ulcer, across the chamber to the other side, the cutting edge being directed forwards; the knife is made to cut its way out, completely dividing the base of the ulcer, opening the anterior chamber, and as a rule causing the escape of the hypopyon. This should be done slowly to avoid the sudden escape of the aqueous, and with it prolapse of the iris and possible damage to the lens.

It is sometimes advisable to keep the wound open for a time in the event of the reappearance of the hypopyon; this may be done by inserting a small probe between the lips of the wound daily.

A recently prolapsed iris may be replaced, but it is generally safer to remove it; it should be seized, freed from the edge of the ulcer all round, drawn slightly forward, and cut off at the level of the cornea. The cut ends will then either retract or may be replaced, but it is seldom possible to avoid adhesions of the iris to the wound.

Sub-conjunctival injections of various antiseptic fluids have been strongly recommended by some surgeons for the treatment of corneal affections, and especially of the different forms of infective ulcer; perchloride and cyanide of mercury have been used most frequently. More recently, however, it has been found that sterilised saline solution, 0·75 per cent of chloride of sodium, injected beneath the conjunctiva is equally useful and less irritating. Enough is injected to produce a moderate distension of the loose conjunctiva all round the cornea; there is a certain amount of reaction after the injection for about twenty-four hours.

This form of treatment has not found much favour in this country, and is now less spoken of even by those who first used it.

In dealing with perforations of the cornea in which the iris is entangled, there is frequently a difficulty in obtaining a firm cicatrix; the involvement of the iris leaving unsound spongy tissue. In some cases a fistula is formed, in others the cornea over the seat of the perforation remains thin and bulged for an indefinite time; the risk of septic inoculation of such an eye is considerable. Complete rest in bed, avoidance of movement and use of the eyes, a compress over the damaged eye, the other one being also bandaged, are means by which sounder union may be secured. Eserine has been recommended to keep down the tension of the eye as much as possible, but there is little or no evidence that eserine reduces the tension except where it has been previously raised.

A method of considerable value in securing firm cicatrisation is the *transplantation of conjunctiva to the seat of the ulcer.*

After freshening the surface of the damaged cornea or the edges of the fistula, this may be done in the following way:—A flap of conjunctiva is dissected up, being left attached by a pedicle; the flap is twisted on itself and tucked into the weakened spot: another method is to dissect up a bridge of conjunctiva at the upper part of the globe, leaving the two ends attached, bringing the bridge down to the seat of the ulcer, and tethering it by one or two sutures applied to the conjunctiva at the edge of the cornea; it may also be done by dissecting up the conjunctiva all round the globe, and bringing it together by continuous suture at its free edge, thus covering the whole cornea.

The conjunctiva seems to act in two ways, partly by the support which it gives to the cornea, and partly by becoming adherent to the seat of the ulcer. The fact of the cornea being covered by conjunctiva does not seem to give trouble, after the sutures come away the conjunctiva falls back again to its proper place.

Oxygen gas has been recommended as a treatment for indolent ulcers of the cornea; the application is made by means of a closely fitting cap, the oxygen being passed over the eye in a constant stream.

Treatment of the Results of Corneal Ulceration.—Opacities.—Little can be done for the treatment of opacities of the cornea. The use of stimulants, like the yellow oxide of mercury ointment, 4 grs. to the ounce of vaseline, or of wine of opium, combined with massage, should be continued for a long time. Opacities due to a deposit of lead on the cornea may be removed by scraping. In some cases undoubted improvement has followed the application of a galvanic current directly to the cornea, but it is unreliable. Tattooing is sometimes done, partly for cosmetic reasons and partly for the improvement of vision. Where the nebula partly covers the pupil, or where an artificial pupil has been made opposite clear cornea, the dispersion of the light passing through the nebula causes much disturbance of vision through the clear part of the cornea; rendering the nebula dark by tattooing it, may result in great improvement of vision. The surface of the nebula is pricked all over by a bundle of needles, and Indian ink rubbed up into a paste is applied to the surface, or a Wecker's grooved needle, containing Indian ink in the groove, is passed into the cornea parallel to its surface, and the ink deposited in the substance of the cornea; the operation has to be repeated several times. Too much should not be done at one sitting; eyes in which the cicatrised cornea is thin, or has the iris adherent to it, should not be touched. The pigmentation of the cornea so obtained is not permanent.

Transplantation of the cornea is not yet within the domain of practical surgery, for though the operation has been done with success as regards the life of the transplanted cornea, its transparency has never been maintained.

TYPES OF *CORNEAL ULCERATION.*—*The Simple Ulcer.*—The simple ulcer may arise from an injury to the epithelium, or from the introduction of a foreign body into the eye. It appears as a somewhat raised gray spot, which becomes a shallow crater with some infiltration of its base; there is circumcorneal congestion, pain, and intolerance of light.

Treatment.—Hot fomentations, compress, and atropine (see general remarks on Treatment).

Infective Ulcer.—The ulcer heals rapidly as a rule, but it may occasionally penetrate deeply, and even perforate the anterior chamber. In this case, instead of clearing, the base and margins of the ulcer become yellow with infiltration, the surrounding cornea becomes hazy, iritis is set up, and a hypopyon forms.

This type of infective ulcer is most commonly seen during the course of a purulent conjunctivitis, but it may occur as the result of an infection of the corneal tissue apart from general conjunctivitis. Its tendency is to penetrate deeply into the cornea, but not to spread widely; in this respect it differs from the serpiginous ulcer.

Treatment.—On the same lines as that of the simple ulcer, but more active measures, such as carefully applied caustics, or the cautery, may be necessary. If purulent conjunctivitis be present its active treatment should be pursued.

Catarrhal Ulcer.—During an attack of conjunctival catarrh we sometimes meet with one or two rounded or crescentic ulcers near the margin of the cornea. They usually heal readily. During the later stages of trachoma, when pannus of the cornea has been formed and the lid has cicatrised, it is not uncommon to see small sharply-cut ulcers with a clear base at the edge of, or on the pannus itself; they generally come with a slight recurrence of irritation in the lids. In addition to the local treatment of atropine the lids themselves require attention.

Serpiginous Ulcers.—There are several kinds of ulcer whose tendency is to spread into the healthy parts in their neighbourhood; in their whole course the character of these ulcers is distinct, the only common feature is their tendency to spread. The main forms are — the acute serpiginous ulcer, the marginal ditch ulcer, the rodent ulcer, and the dendritic ulcer. Some of these are described now, the others will be found in their place later.

The Acute Serpiginous Ulcer; Ulcus Serpens; Hypopyon Keratitis.—This begins as a dirty gray spot showing a loss of surface, with an infiltrated base and edges; the base is uneven, and is covered with broken-down epithelial and corneal cells. It commonly follows a slight injury, and occurs especially in those who have a diseased lacrimal sac. The characteristic feature of this form of ulcer is its advancing edge, which generally appears as a yellow crescentic area situated at one edge of the ulcer. This crescentic area tends gradually to spread over the cornea superficially in one direction, while the part of the ulcer first formed remains stationary or tends to heal. The cornea around the ulcer is cloudy, there is intense circumcorneal injection; a certain amount of iritis is present, the iris is blurred and swollen, enlarged blood-vessels may be seen on it, and posterior synechiæ may be formed. A hypopyon appears very early in the disease. The signs of irritation are very great in most cases, intense pain, intolerance of light, some fever, and a want of sleep; in other cases the signs of irritation are not marked. If not checked the hypopyon increases and may fill the anterior chamber, the ulcer spreads so that a considerable area of the cornea becomes involved, and this either yields to the intraocular pressure and perforates, or the ulcer penetrates deeply and the same result obtains. In some cases perforation is followed by general infection, panophthalmitis, and total destruction of the eye, but, on the other hand, it sometimes marks the onset of a remission of most of the irritative signs, and is followed by a tendency towards healing. Abscess of the cornea is closely allied to the serpiginous ulcer, but it spreads in the substance of the cornea without destroying the surface; if it is seated in the superficial layers the greater part of its anterior wall will break down and form an ulcer, but it may invade the deeper layers and run its whole course without destroying the surface. It is generally not very acute, but it is frequently accompanied by iritis and hypopyon; in some of the very chronic cases the mass of exudation does not go on to active suppuration, but quietly subsides, and is eventually transformed into fibrous tissue.

Treatment.—Fomentations and atropine in the earliest stages, with a compress, followed by the galvanic cautery or the local application of carbolic acid, or some other caustic; Saemisch's operation on the ulcer; the treatment of any lacrimal sac complication (see general remarks on Treatment).

The Marginal Ditch Ulcer; Marginal Ring Ulcer.—This begins as a groove near the edge of the cornea; it is usually rather deep, with sharply-cut edges, with or without infiltration of its base; its tendency is to creep gradually round the edge of the cornea, but it may heal in one part while advancing in another; if not checked the whole cornea may be destroyed by it. It generally occurs in old people with failing nutrition; the cause is not known.

Treatment.—The galvanic cautery should be applied to the advancing edge of the ulcer. Eserine should be used to improve the local nutrition as well as heat and compresses.

The Rodent Ulcer.—A gray infiltration appears at the periphery of the cornea and soon breaks down into an ulcer; the ulcer does not penetrate deeply, but tends to spread slowly and intermittently over the whole corneal surface. The appearance of this form of ulcer is quite peculiar and characteristic. The surface of the ulcerated area is below the level of the rest of the cornea, it is uneven and opaque, and may contain blood-vessels which have spread in from the limbus. At the line of junction with the unaffected part of the cornea, the latter stands up considerably above the ulcerated area, and has abrupt cliff-like edges which are gray in colour and undermined in places. The progress of the ulcer is very slow, and it has periods of quiescence, but it eventually creeps over and spoils the whole surface of the cornea. It occurs chiefly among elderly people, and especially among those who have lived abroad, but it is a very uncommon form of ulcer. The only treatment at all efficacious is to cauterise the advancing gray edge of the ulcer.

Kerato-Mycosis Aspergillina. — This rare affection has been described by several Continental writers, but no case has hitherto been reported in this country. It is due to the growth on the cornea of the fungus Aspergillus fumigatus, which gains an entrance by means of a foreign body or other injury to the corneal epithelium. The fungus appears as a gray mass with a dry crumbling surface, seated generally near the centre of the cornea; the signs of irritation are not severe, and the course of the affection is tedious. The mass of the growth is, as a rule, cast off by exfoliation of the part of the cornea in which it has been growing. Its removal may be hastened by scraping.

THE PHLYCTENULAR ULCER (*Phlyctenular conjunctivitis, Pustular conjunctivitis, Exanthematous or eczematous conjunctivitis, Phlyctenular or pustular keratitis, Marginal keratitis, Herpes of the cornea, Scrofulous or strumous ophthalmia, Lymphatic conjunctivitis*).

This disease appears in a great variety of forms, each of which has different stages; nearly all the forms and stages have received separate names, the result has been the unnecessary complication of a very common affection. The disease is mainly one of childhood; the general type consists in the formation of a limited exudation of lymphoid cells beneath the epithelium of the cornea or conjunctiva; the epithelium breaks down, leaving a small shallow ulcer. The ulcer remains for a time, but is gradually repaired by epithelium creeping over its surface; at the same time a similar ulcer has been formed elsewhere; sometimes the ulcers are very minute, and are known as miliary phlyctenulæ. These are often so numerous as to invade the whole limbus, and even to be scattered over the whole surface of the cornea, which has the appearance of being covered with minute grains of sand. The irritative symptoms are severe, the conjunctiva of the lid is much swollen, there is intense fear of the light and spasm of the lids, the outer canthus is frequently cracked or excoriated, the child shrinks away to the darkest corner and keeps his face deeply buried. At the slightest attempt to open the eyes a copious gush of tears comes out, and it is well-nigh impossible to obtain a view of the cornea. These confluent phlyctenulæ produce a swelling of the limbus, while the cornea in the immediate neighbourhood loses its transparency, and contains a number of small gray points just beneath the epithelium. With small phlyctenulæ, as a rule resolution takes place, the elevations disappear. If they break down into an ulcer by loss of the covering epithelium, the ulcer as a rule heals, but it may become infected and take on the characters of an infective ulcer (see p. 385). In other cases the behaviour is quite different; the ulcer assumes a chronic course and may remain stationary for weeks, vessels grow into the cornea from the limbus towards the crater-like depression, and healing takes place slowly. The healing does not always follow the ingrowth of vessels, the ulcer slowly pushes its way towards the centre of the cornea, carrying the leash of blood-vessels with it. This condition is known as a *Leash ulcer* or *Fascicular keratitis.* The advancing edge of the ulcer is convex towards the centre of the cornea, and somewhat raised in the form of a crescent, consisting of an exudation into the corneal substance, the vessels of the fasciculus ending in the concavity of the crescent. The difference between this leash ulcer and the natural healing of an ulcer by an ingrowth of vessels lies in the fact that the leash ulcer contains a yellow edge of purulent exudation. This condition may last weeks or months, and results in the formation of a band-like opacity which remains visible for years.

Where the ulcers of the cornea are multiple and adjacent to one another the ingrowth of vessels of repair from the limbus may occupy a considerable area of the cornea. These vessels remain as potential blood-channels for a very long time after the complete healing of the ulcer, and are liable to become easily congested, giving rise to a condition known as phlyctenular pannus, or superficial vascular keratitis. The vessels lie beneath the epithelium; at times they are invisible except to careful examination, and at others they are much enlarged, and the corneal surface is rough and uneven.

Causation.—This is a disease of childhood, but it does not occur generally in children under one year old; its most common period is from two to sixteen years; during this time skin eruptions, eczema, and impetigo about the face are very common, together with a similar condition of the nasal mucous membrane. It most commonly occurs when the changes in temperature are great and sudden, as in spring and autumn; as the effect of climatic changes is greater among people who live in damp, ill-ventilated, or overcrowded houses, so this disease is common among the poor, and rarely occurs in children whose surroundings are satisfactory. As a probable result also of environment the children fall into irregular habits of feeding, and eat anything at any odd time instead of having regular meals, whereby the appetite becomes capricious and the desire for unwholesome food is established.

The next most common exciting cause is a previous attack of some acute exanthematous disease, such as measles, scarlet fever, or whooping-cough, by far the most common of which is measles. The eye affection comes on a few weeks after the measles, and is not the same as the conjunctival inflammation which commonly ushers in the first stage of measles. The irritation of head lice is a frequent accompaniment of the disease, and is especially likely to occur, as during this period the children appear sufficiently morose and unattractive to make neglect probable.

It is held by many that this affection is in its nature strumous or scrofulous. The old term Scrofulous ophthalmia, besides including all the eye affections of this group, was applied to all cases in which there was extreme intolerance of light; it was thought that the photophobia was induced by an irritation of the optic nerve and retina, and a consequent reflex spasm of the orbicularis, but inasmuch as treatment applied to the terminations of the fifth nerve in the cornea and conjunctiva lead to diminution of the intolerance of light, we know that the affection is a superficial one only; the intolerance is not greater than may be produced by a foreign body under the upper lid. It is characteristic of all cases of superficial keratitis in the growing period of life that irritation, intolerance of light, and spasm of the lids are much greater than in adult life. Yet the term strumous or scrofulous ophthalmia has this justification, that in a large number of cases of superficial corneal inflammation in the young there is a certain condition of the tissues which makes them favourably disposed to the occurrence of inflammatory outbreaks which are very chronic in their course, and which tend to recur without obvious cause.

This condition is associated with a proneness of the lymphatic tissue in the body to hypertrophy, the glands in the neck or at the angle of the jaw become enlarged, the mucous membrane of the nose becomes swollen and inflamed, and masses of adenoid tissue form in the pharynx. Also if the family history and antecedents of the parents be carefully examined there will be found a liability to tuberculous affections in others of the family. Evidence of this sort has to be received with caution, as it depends on the statements of friends who are often anxious to produce what they think the inquirer is seeking to find. But the liability to recurrence of phlyctenular disease, its association with other lymphatic hypertrophies, the frequency of its appearance in more than one member of a family, point to a peculiarity in the tissues capable of being transmitted from one generation to another, and favourable to the growth of certain organisms. The actual relationship with tubercle is probably no nearer than this: phlyctenulæ have been excised and examined frequently without revealing the presence of anything but staphylococci; tubercle bacilli have never been found.

Treatment.—This should be local and general. Locally a little boric acid lotion, ten grains to the ounce, should be dropped into the eye about three times a day; if there is any photophobia or spasm of the lids atropine should be used night and morning. It is necessary to get a view of the cornea—often a matter of difficulty; this can generally be done by some coaxing; it may be necessary, however, to use lid retractors, or even to give an anæsthetic to get a view. In many of the cases in which the spasm of the lid is greatest the involvement of the cornea is only trivial. It is often possible to obtain relief of the spasm by the sudden shock of cold water falling on the face from a sponge held above the patient; or if the spasm is great and the corneal involvement small, it is useful to evert the lids, dry the conjunctiva, and apply to it freely and to the skin of the lid, especially if eczema be present or the canthus be cracked, a solution of nitrate of silver, mopping up the excess; a few hours after the application the child sometimes spontaneously opens the eyes. When the stage of irritation has passed, yellow oxide of mercury, four grains to vaseline one ounce, may be used for a considerable time.

The fascicular form is very tedious; if the crescentic area is yellow and advancing it must be cauterised, or scraped and touched with carbolic acid, or with the point of a stick of mitigated nitrate of silver.

As a rule it is better not to bandage the eyes, coolness, free access of air are good; to protect the eyes from light, goggles or a shade may be used; bleeding and blisters are useless and harmful; a seton in the scalp is a very active remedy in cases which resist all other kinds of treatment, but it is liable to set up cellulitis of the scalp with suppuration in the neighbouring glands, so that it should only be used in those cases in which all other methods have failed.

As to *general treatment*, it is well to begin with a purge followed by iron, arsenic, or quinine and cod-liver oil or cream as an addition to the diet, regulation of the hours of feeding, the forbidding of cakes or unripe fruit between meals; a change of air to the country or seaside is most beneficial; if this cannot be secured, the child should be made to live in the open air as much as possible. The local skin affections should be treated, adenoids removed, and disinfection of the nose and throat secured.

AFFECTIONS OF THE CORNEA ASSOCIATED WITH THE FORMATION OF BULLÆ.—Herpes of the Cornea.—This affection appears under two forms, simple febrile herpes and the more severe herpes zoster.

Febrile herpes occurs chiefly in children during a febrile attack, such as exanthematous fever, pneumonia, or even a catarrh of the respiratory or nasal passages. During the early stage there is intense photophobia, spasm of the lid, and lacrimation; one or more minute blisters form on the surface of the cornea, and when seen some of them may be entire, containing fluid, or more generally they have burst, and we see a shred of epithelium attached to the edge of a shallow pit in the cornea. The bullæ may be arranged in line, or they may form a constellation in one part of the cornea; the edge of the pit is quite clear and sharp, and appears as if a piece of the cornea had been cut out by a spud or finger-nail. The healing generally takes place as in an ordinary ulcer after the shedding of the epithelium. The sensibility of the cornea is said to be lowered in the region of the blisters, and the tension is also said to be reduced; it is difficult to apply satisfactory tests in the condition of irritation present, but the tension is not always lowered, and the cornea is sometimes hyperæsthetic; these may vary in the different stages of the affection. Febrile herpes is only characteristic in the earliest stage, when it is not often seen; at a later period it cannot be distinguished from a simple or phlyctenular ulcer.

Treatment.—Rest, shade for both eyes, atropine, and general treatment of the catarrhal state, are measures generally enough to heal the ulcer without leaving a mark, but it may become infected like any other ulcer (see general remarks on Treatment of Ulcers of the Cornea.)

Herpes zoster of the cornea occurs as part of herpes zoster ophthalmicus, an inflammation of the first division of the fifth nerve or Gasserian ganglion. Where the supra-orbital or supra-trochlear branches only are affected the cornea as a rule escapes, but when the nasal branch is involved, shown by the spots occurring on the tip of the nose, there is generally ulceration of the cornea, iritis, and sometimes cyclitis; the nerves of the front of the eye are derived from the nasal branch of the fifth nerve by way of the long root of the lenticular ganglion. The herpetic attack is preceded by intense neuralgia, pain and swelling of the lid; this latter is so great that the affection is often thought to be erysipelas; one or two vesicles appear on the cornea and form ulcers; in spite of the intense pain the cornea is as a rule completely anæsthetic, and may remain so for a long period.

The *treatment of* the ulceration is that of a simple ulcer, but it must be borne in mind that the cornea is insensitive and cannot protect itself against foreign bodies and other kinds of injury, so that it must be bandaged and carefully watched.

The dendritic ulcer is a well-defined variety of superficial corneal ulceration. It is generally seen as a groove in the cornea consisting of a central stem with small buds or branches at each side; the ulcer has a tendency to creep over the cornea and spoil a large part of the surface. The ulcer sometimes occupies the centre of the cornea, but in the most characteristic cases extends from the limbus as a broad trunk which gives off branches on each side, the branches themselves ending in secondary branches or buds. In its earliest formation the affection is herpetic in character, the buds have the appearance of small bullæ. The appearance of the dendritic ulcer is very striking and beautiful after staining with fluorescine.

It is not known what is the cause or exact nature of the dendritic ulcer, but it probably belongs to the herpetic group; it occurs frequently in people who have been subject to malaria, and has been described as *malarial keratitis*, but in this country it is seen not seldom in patients who have never been abroad and who have never had any form of malaria. It occurs in those who are past the best period of life, or who are temporarily run down; I have seen it twice in young children, both of whom were much exhausted by prolonged joint suppuration.

Treatment must be radical. The course of the ulcer is rarely stopped by the ordinary routine measures, such as fomentations, atropine, compresses. The galvano-cautery at a very low temperature may be used, but there is risk of producing a permanent scar with it; carbolic acid is safer and better, but several applications are often needed. The most effective treatment is absolute alcohol applied by dipping a small roll of lint into the alcohol and rubbing the ulcerated surface with it; the epithelium for some distance around the ulcer is removed, but it is quickly regenerated. The only drawback to this very efficient method, which we owe to Swanzy, is its painfulness.

An affection closely allied to febrile herpes is the *superficial punctate keratitis* (Fuchs). A number of small gray elevations appear in the cornea arranged like febrile herpes in lines or constellations; their number may vary from ten or twenty to about a hundred; gray lines are also seen in the cornea, which are probably caused by exudation into the corneal nerve trunks. There is much photophobia, although the cornea is somewhat anæsthetic. The attack is part of a general catarrhal condition affecting the parts supplied by the fifth nerve; the irritation clears up in a short time, but the gray spots may remain for months.

Local Treatment. — Protection from light, and atropine (see general remarks on Treatment).

Recurrent Bullous Keratitis.—A solitary blister sometimes forms spontaneously under the corneal epithelium, attended by severe neuralgia, intolerance of light, and watering. Sometimes there has been an injury at the spot, and for a time recurrences take place in the same eye, but the affection is often independent of injury, and the relapses occur either at definite times in the year or at intervals depending on some variation in health; in some cases the attack always occurs at a menstrual period, though not at each one.

Treatment.—Bandage, rest, atropine, sometimes tearing off the shred of epithelium left by the bulla, causes considerable relief of pain. Quinine has been advised as a prophylactic, on the assumption of a malarial basis, but it is not possible to predict the onset of an attack.

Bullæ are sometimes formed in eyes blind from old glaucoma, in which the cornea is more or less devoid of sensation. They also occur upon old leucomata associated with a temporary increase of tension; it may be necessary to do an iridectomy for the cure of the increase of tension if the eye is worth saving.

Filamentary keratitis is a very rare disease. Numerous small threads are seen hanging from the surface of the cornea and attached to it quite firmly. The threads consist of a twisted strand of epithelial cells having a bulbous end, and covered in places by mucus. When the threads drop off the base heals, but their place is taken by others caused by degeneration of adjacent epithelium. The treatment consists in rest, atropine, and bandage.

KERATO-MALACIA.—Infantile Ulceration of the Cornea.—This serious affection occurs in young children who are the subjects of grave disturbance of nutrition, due either to starvation or improper feeding, or to the exhaustion of disease. The first manifestation of the affection is a dryness of the conjunctiva and cornea, and the formation of a small triangular foam patch at the inner and outer margins of the cornea in the exposed part of the eye. This lack of lustre or dryness, which varies greatly in degree, is due to a fatty degeneration of the epithelial cells, and a consequent inability of the tears to moisten the surface.

Local infection of the cornea through the degenerated epithelium is followed by deep infiltration and rapid destruction of the whole or part of it; the actual organisms present have been found to be streptococci or pneumococci, and the foam patches contain the xerosis bacillus.

During its whole course the disease is attended by very slight local symptoms, little or no discharge, and scarcely any intolerance of light. Kerato-malacia is rarely present without there being grave disturbances of general nutrition; the children, if not actually wasted, have an unhealthy, ashen colour; they are, however, for the most part greatly emaciated by constant diarrhœa and vomiting, and in spite of every care they die.

Kerato-malacia appears in England mostly in hand-reared infants badly nourished in consequence of unsuitable food or of prolonged diarrhœa. Poorness or insufficiency of milk, the use of condensed milk from which the cream has been separated, the use of patent starch foods in the place of pure milk, are among the most common causes, but it may occur in hand-reared infants, in the feeding of whom the nutritive value of the milk has been impaired by prolonged or repeated boiling carried out with the laudable desire of rendering it sterile. One is reminded of the experiment of Majendie, who fed a well-nourished dog on nothing but pure white sugar and water, with the result of producing a central ulcer of the cornea. Severe exhaustion after acute fevers or pneumonia is a predisposing cause, and is often seen in the late stages of mild ophthalmia neonatorum in babies suffering from congenital syphilis. In countries where religious fasting is strictly observed it also occurs in breast-fed children.

Treatment, both local and general, should be directed towards improvement in nutrition. Warmth by compresses and hot fomentations frequently applied; the local use of eserine in the form of an ointment, one grain to the ounce three times a day, are the best local measures, while general treatment should be in the direction of securing proper food, milk diluted according to age, cream or cod-liver oil, and meat juice, and the diarrhœa should be combated.

ULCERATION OF THE CORNEA FROM EXPOSURE.—When the cornea is exposed to the air it becomes rapidly dry, and if not moistened by tears its surface is destroyed and a way provided for the entrance of organisms.

This condition is produced by cicatrisation and contraction of the lids from old injury or disease; it occurs in facial paralysis when the lower lid no longer keeps in position; also in the extreme protrusion of the eye sometimes met with in exophthalmic goitre, and in orbital tumours. It is also seen during the condition of apathy patients suffering from exhausting disease.

Treatment.—The eye should be closed temporarily by a piece of strapping, but if the exposure is likely to be permanent, the edges of the lids may be pared and stitched together for a certain distance.

NEURO-PARALYTIC KERATITIS.—When the fifth nerve is paralysed the cornea becomes anæsthetic and is readily exposed to injury; foreign bodies are not swept away, and the surface is no longer kept moist by the movements of the lids induced by the sensations of the cornea. The epithelium becomes cast off from a portion of the surface, and an ulcer is formed; this form of ulceration runs a very chronic course, and is little affected by treatment; infection may take place, and the eye may be lost. The changes have been described as beginning interstitially without loss of epithelium.

It is held by many that the paralysis of the fifth nerve is associated with a trophic disturbance in the cornea, and although the results of section of the fifth nerve on the nutrition of the cornea tend to show that if the cornea be protected, such section has no influence on it, yet if we take the analogy of other parts, *e.g.* the glossy skin which occurs in paralysis of sensory nerves, it is highly probable that the fifth nerve does exert an influence on the nutrition of the cornea. Clinical evidence is in favour of this view.

The *treatment* is that of ulcerative keratitis, especial care being taken to secure protection of the eye.

INTERSTITIAL OR PARENCHYMATOUS KERATITIS.—In this disease the deeper layers of the cornea are attacked by chronic inflammation with the formation of opacities and with the development of blood-vessels. The changes in the cornea show a tendency to clear up for months after the inflammatory symptoms have subsided; there is no tendency to ulceration or suppuration.

The appearance of the changes in the cornea is preceded by circumcorneal congestion, lacrimation, and intolerance of light; an examination of the eye is made difficult by spasm of the lids, hence in many of the worst cases it is not possible to say what is the exact condition of the cornea. The changes show themselves in many forms, but the most common mode of onset is the appearance at the periphery of the cornea in one spot of a group of maculæ deep in the corneal substance; the epithelium covering them becomes swollen and loses its lustre, so that the maculæ are only seen dimly. At the same time the vessels of the limbus become much enlarged at this spot, and the limbus itself is swollen and encroaches somewhat on the surface of the cornea in the form of a vascular crescent; this encroachment does not exceed certain narrow limits. At the same time, there is an ingrowth into the corneal substance of vessels coming from the deeper layer of the sclerotic, appearing beneath the limbus and having no connection with its vessels. These are long and thin, and are in the substance of the cornea; they have the appearance of long tufts, and have been likened to the fibres of a besom; they gradually force their way into the substance of the cornea, the area of maculæ still extending in front of them towards the centre; sometimes the process starts from two opposite sides of the cornea, the skirmishing line of maculæ advancing towards the centre till they meet those coming from the other side. In another group of cases, instead of starting peripherally from one or two points, the maculæ make their appearance towards the centre of the cornea where they become confluent, and from this region gradually extend towards the margin; the cornea is almost uniformly gray, the centre being more opaquely gray than the periphery. At the same time the epithelium is swollen and loses its polish; with a magnifying glass or corneal microscope the individual cells can be seen unevenly œdematous. There are varieties in the amount of vascularity present, in some cases the vessels can be seen singly, in others they are very numerous and appear only as a pink patch in the cornea, a condition known as a *salmon patch.* The density of the opacity varies greatly, and is, as a rule, patchy; at the height of the attack the deeper parts of the eye cannot be seen with any clearness, but, as a rule, it can be made out that the anterior chamber is deep; that the pupil is either not dilated freely, or that adhesions are present; and that there is a deposit on the back of the cornea, *keratitis punctata,* the sign of involvement of the ciliary body. The tension of the eye is generally rather lowered. In older patients from the beginning the corneal opacity in its earliest stage sometimes takes on the striated form, gray lines are seen deep in the corneal substance, these are followed by the development of a general haze of the deepest layers of the cornea.

Interstitial keratitis is always chronic in its course, the opacity of the cornea and the inflammatory and irritative symptoms go on increasing for about two months, and then slowly decline. At its worst the vision is much reduced even to the perception of hand movements. As the attack abates the ciliary congestion diminishes, the cornea clears from the periphery, and the sight begins to improve, the clearing and improvement in vision continuing for many months. The severity of the attacks varies very greatly; in young children it is not uncommon to see a few maculæ at the periphery, or a slight central haze with ciliary congestion disappearing entirely in a very few weeks, an attack in miniature only; on the other hand, in the worst cases the irritative symptoms may last twelve months or longer, and the cornea may be converted into a dense white tendinous structure absolutely opaque. It happens rarely that the cornea yields owing to inflammatory softening and becomes bulged; this is sometimes uniform, and may subside without causing any gross change in the corneal curvature; at other times the yielding of the cornea may be preceded by the formation of a gumma of the cornea, a local mass of granulation tissue surrounded by a dense felt of vessels, and having a tendency to degenerate at the centre. It is very uncommon for an abscess or ulcer to form; the gumma, like most of the manifestations of interstitial keratitis, undergoes slow involution in most instances.

The participation of the uveal tract of the eye in interstitial keratitis is one of the marked features of the disease. It is difficult to form an estimate of the depth in the cornea of the changes even with a highly magnifying lens, especially as in the period of evolution the irritative symptoms prevent careful examination, but the deposits in Descemet's membrane are generally well-marked dots of varying size, sometimes densely crowded and very fine, at other times large, like spots of grease; these are either scattered generally over the back of the cornea, or chiefly collected at the lower part. Occasionally the appearances suggest an almost universal change in the endothelium in certain areas of the lower part of the cornea, as if there were an irregular deposit of amorphous cretaceous granules on it.

Iritis is shown by hyperæmia of the iris, posterior synechiæ, and exudations into the pupillary area; the failure of the pupil to dilate under atropine is sometimes marked, even when on recovery there can be found little or no sign of past iritis: the failure of dilation probably arises from the absence of absorption of the atropine through the inflamed cornea. After recovery it is often possible to make out recent extensive peripheral choroiditis, either in the affected eye or its fellow. The share taken by the uveal tract varies from a degree in which it can scarcely be verified to almost pure irido-cyclitis, with slight involvement of the cornea only.

A rule, with scarcely any exception, is that the disease occurs in both eyes; the interval between the two, however, may be as much as from three to four years; recurrences may take place, but after a typical well-developed attack they are rare. Among young children it is not uncommon to see slight attacks of interstitial keratitis or hybrid interstitial and phlyctenular attacks recurring several times, followed at a somewhat later period in life by a well-developed attack of interstitial keratitis, after which there is no more trouble.

It is well to warn the patient's friends, as soon as the nature of the affection is recognised, of its probable duration, of the almost certain involvement sooner or later of the other eye, and also of the ultimate probable recovery of very useful vision. Nothing is so encouraging to the patient as the assurance of recovery during the long weeks of suffering.

Causes of Interstitial Keratitis.—It is more common in females than in males; the age of greatest prevalence is from about seven to eighteen, but these limits are not very strict. I have seen a few cases in quite young children under the age of two, and it is not uncommonly seen up to twenty-five, and sometimes up to thirty years.

The most frequent cause of interstitial keratitis is hereditary syphilis, which in most of the cases may be recognised by its stigmata without having to question the parents directly. But evidence of repeated miscarriages, or of premature births, or of great mortality among the children in early life, may be easily obtained, as also the testimony as to infantile skin eruptions or snuffles.

The signs by which hereditary syphilis may be recognised are by the shape of the cranium, the physiognomy, the teeth and the choroid.

The frontal eminences are prominent and rounded; the central part of the face is depressed owing to the sunken bridge of the nose, and to the flattening of the front of the superior maxillary bones. The mouth itself shows linear cicatrices radiating outwards especially from the angles, and frequently depressed cicatrices are seen in the skin of the neighbourhood. Shotty lymphatic glands may be felt in the neck, and the throat shows signs of old ulceration. An examination of the choroid of the other eye by the ophthalmoscope will often reveal the scars of old disseminated choroiditis.

The appearance of the teeth is one of the most characteristic signs of inherited syphilis: in the milk teeth there is nothing characteristic, but the incisors very often become carious. In the permanent teeth attention should be directed to the upper central incisors, although the others may also show signs. In the upper central incisors two main types are met with, those in which the cutting-edge is narrowed and has a central notch in it, and those in which the notch is not present, but the cutting-edge is much narrower and thinner than the crown of the tooth, the screw-driver type. The teeth are also undersized and separated from one another by spaces.

Other manifestations of inherited syphilis are deafness from internal ear disease, periosteal nodes, or effusions into the knee-joints.

Interstitial keratitis also occurs rarely in acquired syphilis, its course is much the same as in the hereditary disease.

Cases occur occasionally in which the most searching examination of the patient fails to reveal any of the signs of hereditary syphilis, and in which no evidence in favour of it can be obtained from the family history. Such patients are frequently in bad health and are rapidly losing flesh ; it is not possible to demonstrate the presence of tubercle in such cases, but the probability of it is great, especially where there are nodular growths springing from the angle of the anterior chamber, as sometimes occurs in such cases. Microscopically nodules very suggestive of tubercle have been found in the angle of the anterior chamber, and the tubercle bacillus has been identified in the cornea in one case by Zimmermann.

The opportunities for examining by the microscope an eye in the acute stage of interstitial keratitis have been very rare. Such an examination shows a cellular infiltration of the posterior half of the cornea increasing in density as the posterior elastic lamina is approached ; at this point the cellular infiltration is so great that the cornea appears to consist entirely of round-celled exudation ; its proper structure is hidden. Descemet's membrane is thrown into folds, and there are deposits of round cells upon the endothelium. Newly-formed blood-vessels are present in the deeper layers of the cornea. The anterior layers appear normal. The round-celled infiltration also invades the ligamentum pectinatum, the iris, and ciliary body.

Treatment.—Much may be done to alleviate pain and distress in interstitial keratitis. Protection from light should be secured by dark goggles, or a shade made to cover both eyes. Heat, by means of hot compresses or fomentations, is occasionally very useful in reducing the discomfort of the patient. Locally we should carefully avoid the use of any kind of irritant as long as the disease is advancing. Atropine, 4 grs. to the ounce of vaseline, should be used to prevent or limit the effects of iritis, and its use should be maintained until the height of the disease is well past. When the symptoms have begun to abate, irritant or stimulant remedies may be begun, the chief among which are the yellow oxide of mercury ointment, which should be begun cautiously in a strength of four grains to the ounce. Calomel may be flicked into the eye, or hot steam applied, or wine of opium dropped in. If there is any return of irritation their use must be discontinued. The cornea may be further stimulated to clear by massage combined with the use of the yellow ointment. This treatment may be continued for months or years.

As a rule most cases do not require bandaging, but if there is a probability of the cornea yielding to pressure it must be supported.

In the *constitutional treatment* mercury should be used, the method by inunction being perhaps the most satisfactory for prolonged use, or mercury with chalk may be given, care being taken that the patient be not salivated.

As the patients are often anæmic and much depressed, this treatment should be combined with syrup of the iodide of iron and cod-liver oil; at the same time general hygienic measures, warm clothing, good food, and fresh air should be secured. In any case the results of treatment are not brilliant, the disease appears to run its course unchanged and to invade the second eye while the patient is still under treatment for the first. However, there is something to be done by constitutional treatment; from observation of cases at the Victoria Hospital for Children during a series of years, I found that the cases which were treated by mercury ran a shorter course, and had less tendency to recur than those not so treated, and also that the complications, such as extreme deafness, were more frequent in those not under mercurial treatment.

LOCALISED INTERSTITIAL KERATITIS OF ADULTS (Keratitis profunda).—A gray opacity appears in the deeper layers of the cornea near its centre; it consists of maculæ or striæ; sometimes the centre of the opaque area is comparatively clear, the opacity forming a not very dense ring round the centre; the striæ are generally seen in the earlier stages, and when the opacity is fully developed they disappear. There is frequently deposit on the back of the cornea, and evidence of iritis or irido-cyclitis, and vessels may grow into the cornea from the periphery. At its height the condition of the eye much resembles the interstitial keratitis of hereditary syphilis. There is generally not a great amount of irritation; the attack lasts a month or six weeks, and passes off, leaving a certain amount of permanent opacity. The patients are generally beyond middle life, and only one eye is as a rule attacked.

The causes of the disease are obscure: it has been ascribed to cold, to rheumatism, to malaria; many of its subjects suffer from defective terminal circulation, cold hands and feet, dyspeptic troubles, and constipation. The local *treatment* should be protective glasses or a bandage; hot fomentations and atropine, 4 grs. to the ounce of vaseline; the general treatment should be in accordance with the requirements of each case.

STRIATED KERATITIS.—The appearance of gray lines in the corneal substance occurs in a variety of conditions; delicate gray lines are often seen extending at right angles to a wound in the corneal substance, whether the whole cornea has been cut through or not; they are sometimes present extending from the area of a deep ulcer; they occur in some cases of iritis and irido-choroiditis, and are not unfrequently the earliest form taken by the opacity in interstitial keratitis; they are sometimes double-contoured, suggesting a tube containing an opalescent fluid. They are said to be sometimes caused by a compress which has been worn for a long time. Their most common appearance is after cataract extraction, extending from the wound at the upper periphery of the cornea downwards into the substance of the cornea; they are more common where the cornea has been much bruised during the operation, but a certain amount of striation is present after nearly every extraction.

Striated keratitis is at most a temporary phenomenon; it either disappears entirely after a short time, or gives way to some more permanent general haziness. The explanation of the occurrence of the lines is not simple; they probably have more than one cause; there is no structure in the normal cornea which corresponds with them, but certain tubes can be made to appear in the cornea by injecting between its lamellæ mercury or air under pressure, Bowman's tubes. Some of the opaque lines are to be explained by the natural formation of Bowman's tubes, by the pressure of fluids in inflammatory conditions of the parts adjacent to the cornea. They have been formed experimentally by making sections of the cornea in rabbits, and have been then found to be caused by wrinkling of Descemet's membrane; the kind which follows cataract extraction may have this cause.

Leprosy sometimes attacks the cornea; nodules form generally at the margin of the cornea, associated with iritis. They generally break down, leading to loss of the eye; the cornea is also sometimes anæsthetic.

Sclerosing Keratitis.—A relapsing, persistent, subacute inflammation of the ciliary region involving the adjacent part of the cornea, and sometimes attended by iritis. The recurrences generally occur in fresh places, and the general effect of each attack is to leave conical or triangular areas of opacity at the margin of the cornea with the base directed outwards; these areas are sometimes very dense and blue-white in colour, and approximate in appearance to the sclerotic.

The cause is unknown. The local treatment should be rest, fomentations, and atropine.

Calcareous Film of the Cornea (Transverse Calcareous Band).—This is a form of degeneration which occurs in the superficial layers of the cornea, in the shape of a broad band or oval patch of dirty gray colour lying in the part of the cornea that is commonly exposed. It lies just beneath the epithelium, and consists mainly of fine calcareous granules united with hyaline substance. Its development is very slow; it starts on each side of the cornea, and gradually progresses towards the centre.

It occurs mostly in eyes that have been lost from old irido-cyclitis, but it is occasionally met with in old people whose eyes are otherwise healthy.

Treatment.—If the eye is otherwise good the film may be removed by scraping and afterwards treating the cornea as if it had been injured.

Congenital Opacities of the Cornea.—In one variety of congenital opacity the cornea appears to have the structure of sclerotic, the opaque sclera seeming to extend for a certain distance into the cornea, and only gradually thinning into clear cornea. Such eyes are often microphthalmic; the condition may be seen in more than one member of the family.

In other cases a fine ring of opacity is seen just inside the corneal margin; it has the same appearance and situation as the arcus senilis (arcus juvenilis).

Other opacities are due to fœtal inflammation, such as interstitial opacities of various kinds. In congenital hydrophthalmos the whole cornea is sometimes faintly milky.

The arcus senilis is a non-inflammatory degeneration of the cornea, appearing as a gray line at the upper and lower parts, and sometimes meeting at the outer and inner; it is densest above and below, and is separated from the sclera by a thin band of transparent cornea. The gray line is due to a hyaline change in the tissues of the cornea, and has no significance.

Blood-staining of the cornea is found when the anterior chamber has been filled with blood for a long time. The cornea appears to be of a red-brown colour, and is quite opaque; the absorption goes on very slowly from the periphery. The staining is due to deposit in the cornea of blood crystals which have formed after diffusion of the blood colouring matter through Descemet's membrane.

Keratitis Punctata.—This term has already been used; it is commonly applied to the deposits on the back of the cornea that are met with in inflammations of the ciliary body. A number of fine dots is seen on the back of the cornea, sometimes evenly scattered, but more often found on a triangular area at the lower part of the cornea where they are formed by deposition. The dots consist of shreds of fibrin and leucocytes cast off from the ciliary body and deposited on the corneal endothelium. Where they are not soon removed by absorption they produce destructive changes in the endothelium. Some of these dots are of large size, and have the appearance of spots of grease on the back of the cornea. It is possible that these deposits may increase in size; when examined in the fresh state they have been found to contain colonies of bacteria (Snellen).

Conical Cornea.—The central part of the cornea begins to bulge very gradually without inflammatory symptoms, forming a blunt cone; in the more advanced cases the conicity is readily seen by looking at the profile of the cornea. The disease makes itself felt by a disturbance of vision, objects can only be seen clearly when held close to the eye, yet the sight is scarcely improved by concave glasses. The slighter degrees may be recognised by the ophthalmoscope by a shadow seen on the background of the eye, crescentic or circular in shape, and shifting with the movements of the mirror; the condition may also be recognised by examining the corneal reflex with a Placido's disc, and comparing it with that from a normal cornea.

The disease may come to an end spontaneously, or in the worst cases a nebula may be formed at the apex of the cone which is rather below the centre of the cornea; perforation never occurs.

The affection is a rare one; it attacks both eyes in young adults, especially women. It is atrophic in nature, and is due either to some developmental peculiarity in the corneal tissue, or to defective nutrition in the part farthest removed from the blood-vessels.

Treatment.—In the early stages some improvement may be effected by prescribing concave glasses with cylinders, the proper strength of which may be found out by the shadow test; these may be used either alone or combined with an opaque screen having a small hole or narrow slit cut in it. Vision has been improved in many cases by hyperbolic glasses as suggested by Ræhlmann.

Of other methods of treatment eserine or pilocarpine used continuously has been said to reduce the conicity, but in most cases it is of no use.

Operative measures, with the object of flattening the cone by forming a resistant cicatrix at or near it, are to be recommended where the impairment of vision is very great.

The apex of the cone may be removed by shaving it off with a Graefe's knife without opening the anterior chamber, and after two days touching the raw surface with mitigated nitrate of silver stick. An elliptical flap may be removed from the apex of the cone, and the cornea afterwards sutured; a small disc of the outer layers of the cone may be separated by a Bowman's trephine, and the surface allowed to heal and contract. Multiple punctures may be made into the apex of the cone with a fine needle, and repeated after some weeks, or the galvano-cautery may be used at a very dull heat to burn the surface layers of the cornea; if perforation take place during burning no harm will result to the deeper parts if care be used, as the cautery is at once chilled by the escaping aqueous. This last method has the advantage of being free from the risk of infection.

Hydrophthalmos (Buphthalmus).—The cornea undergoes general and progressive enlargement, and sometimes becomes hazy in this affection. The enlargement is part of a general enlargement of the globe, and may be looked upon as congenital or infantile glaucoma.

Tumours of the Cornea.—Tumours of the cornea are rare. Myxomata, fibromata, sarcomata have been described as well as epitheliomata starting from the limbus, and cysts following injuries, formed by the inclusion of cells from other parts in the corneal substance.

INJURIES.—*Foreign Bodies in the Cornea.*—Foreign bodies, fragments of steel or iron, small pieces of stone or particles of ash from locomotive engines, imbedded in the cornea are very common accidents, and give rise to varying amounts of pain and irritability.

They sometimes remain for long periods without exciting disturbance, as among those engaged in metal grinding, in whom the cornea may often be seen studded with minute fragments of stone which have been there an indefinite time. Scales of seeds or the wing cases of small insects may adhere to the cornea by their concave surfaces.

Particles of steel or iron become partly oxidised, and the foreign body may be often seen surrounded by a brown ring, which consists of sodden epithelium impregnated with oxide of iron. This ring may remain behind after removal of the particle, and continue to keep the eye irritable. If not removed, foreign bodies are generally cast off by exfoliation of the part in which they are lying; during the whole of this time the eye is liable to infection at the seat of the injury, especially where the lacrimal sac is not healthy.

For their removal the eye should be thoroughly cocainised, two drops of a 2 per cent solution of cocain should be put into the eye three times at intervals of a minute; the patient should be seated facing a light, the surgeon standing behind him. The lids being held open by the left index and middle fingers, the foreign body may be removed, at first by the edge of a piece of clean blotting paper, or if it is imbedded, by a spud or needle. The ring of oxide round the foreign body should also be removed. If the anterior chamber be penetrated other measures must be taken to prevent the object from being pushed into the chamber, or the parts beneath from being injured. If of iron or steel the attempt should be made to remove it with a strong magnet; this is often unsuccessful owing to complete oxidation of the metal.

Eserine should then be used, one or two drops of a solution of 2 grs. to the ounce, to contract the pupil and protect the parts beneath from injury. A keratome or broad needle should be passed into the anterior chamber and held against the back of the cornea behind the perforated spot by an assistant. This will prevent the foreign body being thrust into the chamber by the attempts at removal from the front of the cornea which must now be continued.

After removal of any foreign body in which the corneal surface has been broken, a compress should be kept on till the wound is healed, and atropine should be used as for the treatment of a corneal ulcer; but where the anterior chamber has been opened the atropine should not be used until it has become closed.

Sometimes the track of a perforation made by a particle of metal can be seen in the cornea without any trace of the object itself. The iris should be examined carefully, the pupil should then be dilated, the lens minutely inspected, and the fundus systematically examined by the ophthalmoscope. Finally, in cases of doubt or difficulty the X rays may be used to determine the presence or absence of a foreign body.

Wounds of the cornea made by blunt or jagged instruments are often irregular, and their edges will not come into apposition. If the iris is protruding, the exposed part should be drawn slightly forward free from the edges and cut off, the edges of the cut iris should then be tucked back. It is not advisable to attempt to return to the interior of the eye a piece of iris which has been bruised in the wound and exposed to septic contamination. A very useful measure in closing gaping wounds of the cornea is to dissect up a flap from the adjoining conjunctiva and place it over the wound in the cornea after thoroughly washing the parts with an irrigator.

Burns.—After scalds by hot water, or burns by acids or alkalies, the cornea looks steamy and dull. The extent of the injury depends to some degree upon the nature of the agent effecting it; alkalies, lime, or sulphuric acid produce serious and permanent damage, whereas even strong nitric acid or blistering fluid may effect only temporary damage. In the worst cases the cornea appears dry and white, and is completely anæsthetic; this condition is likely to end in complete necrosis. Lime burns are among the most common injuries, but fortunately the lime is nearly always partially slaked; the full effect of lime or caustic alkalies is produced very slowly, and only as recovery takes place can the amount of cicatrisation be appreciated. In every injury of this kind the prognosis should at first be strictly guarded even where the change appears to be slight only; owing to cicatricial changes the conjunctiva may be drawn more or less over the cornea, or adhesions may form between the lid and the cornea.

Treatment.—All trace of the caustic agent should be removed; treatment should be by rest, a compress and atropine, a few drops of a solution of 2 grs. to the ounce once a day. The contractile cicatrisation should be prevented as far as possible by putting in pure castor oil three times a day, and once a day allowing free movement of the eye in all directions, carefully separating the lid from the eye.

Injuries of the Cornea.—Superficial abrasions are extremely painful, and cause much watering hyperæmia and intolerance of light owing to the friction of the lid against the denuded surface. These injuries are frequently caused by branches or leaves of trees, or by the finger-nail. The loss of surface is generally confined to the epithelial layer, and is not easily seen unless we examine the corneal reflex.

Treatment.—The firm application of a compress and atropine, in the form of an ointment (4 grs. to the ounce); cocaine gives temporary relief, but the prolonged use of it is likely to produce softening of the epithelium. As sleep is often impossible it may be advisable to give a hypnotic. Rest and immobility of the eye for a few hours are enough to secure regeneration of the epithelium.

Relapses of abrasion are sometimes seen without any fresh injury (see "Relapsing Bullous Keratitis").

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Corns. *See* SKIN, BENIGN TUMOURS.

Corrosive Sublimate. *See* TOXICOLOGY.

Coryza. *See* NOSE.

Cough.—Cough is an important symptom in many diseases, and may constitute the chief complaint of the patient, while in other diseases the presence and character of a cough hardly noticed by the patient may give important aid in diagnosis.

The Act of Coughing.—Coughing is mainly an expiratory act in which air is expelled explosively through the mouth. Air is drawn into the chest and shut in by the closure of the glottis. The intrathoracic tension is increased. The gates of the larynx are then opened, and the imprisoned air escapes explosively. The gates of the larynx are the true and the false vocal cords. It is by the apposition of the false vocal cords that the intrathoracic tension can be increased to a sufficient degree to permit of an explosive cough. In animals, such as the sheep, in which the false cords and the ventricles of Morgagni are undeveloped, there is no proper explosion on coughing.

Cough may be voluntary, but is usually reflex. When the irritation is not too strong, the impulse to cough can be voluntarily restrained.

The impulse to cough commonly results from irritation of some of the branches of the vagus, and may proceed from—

1. The respiratory mucous membrane (naso-pharynx, larynx, trachea, bronchi).
2. Aural irritation, as from foreign bodies in the external meatus, or rarely from chronic otitis.
3. The back of the tongue, through the lingual branch of the superior laryngeal nerve.
4. Gastric irritation—the "stomach cough" which occurs in association with chronic dyspepsia both in children and adults.
5. Irritation of branches of the vagus by enlarged bronchial glands, or by enlarged glands, tumours, or abscesses in the mediastinum.
6. The skin, especially of the throat and chest.

Apart from local irritation, cough may be purely "nervous."

THE CLINICAL VARIETIES OF COUGH.—*Diseases of the Larynx.*—In simple and in membranous laryngitis the cough is commonly hoarse, barking, and imperfect, or, as is usually said, croupy. In ulceration or thickening of the mucous membrane of the larynx the cough may be husky and imperfect, or may be loud and clanging.

When a malignant tumour or an aneurysm presses upon the trachea, or involves the recurrent laryngeal nerve, the cough is commonly loud, resonant, and brassy, and has been compared to the cry of a gander.

In paralysis of both vocal cords the patient is unable to cough; in abductor paralysis, whether unilateral or bilateral, the cough is unaffected; in adductor paralysis there is perfect cough combined with aphonia.

Diseases of the Lungs.—In *bronchitis* the cough is at first short and dry, and may be painful; as secretion increases it becomes moist, and may be paroxysmal. Severe paroxysmal cough may be present in *fibroid phthisis*, and, more characteristically, in *bronchiectasis*. In *phthisis* cough is one of the early symptoms. At first it is dry and hacking; later it becomes loose, and is attended with muco-purulent or purulent expectoration. When cavities develop the cough may become paroxysmal, and is often most severe in the morning. Occasionally advanced, and even advancing, phthisis may be present, with entire or almost entire absence of cough. This occurs especially in lunatic patients. In *pneumonia* the cough is frequent, short, dry, restrained, and associated with severe pain in the side. In acute *pleurisy*, cough is usually, but not always, present. It is seldom so distressing as in acute pneumonia.

The paroxysmal cough of *pertussis* is very characteristic. A series of coughs follow each other so rapidly that no inspiration can occur. The patient becomes cyanosed, the veins of the forehead and scalp become distended, the eyes become prominent, and the tongue is often protruded. Bleeding from the nose may occur. At last a sudden inspiration takes place with a loud whoop. This may be followed immediately by a second or by several series of coughs and whoops before the paroxysm is over. At the end of the paroxysm a quantity of mucus is generally expelled with some violence, and in young children vomiting is usual. During the paroxysm the child seizes hold of its nurse or of some piece of furniture for support, in order to give purchase to the accessory muscles of respiration. In pertussis the cough is apt to be most troublesome at night.

Enlarged glands at the root of the lung may give rise to very severe and persistent coughing, sometimes paroxysmal in character, either by pressure on a bronchus or by irritation of branches of the vagus. When symptoms of pressure on a bronchus are absent it is often difficult to make out the cause of the cough. Eustace Smith has called attention to a physical sign often met with in these cases. If the child is made to throw the head as far back as possible, a bruit is heard on auscultation over the manubrium sterni which disappears when the head is brought forward again. He attributes the murmur to pressure on the left innominate vein.

It must never be forgotten that the irritation of the pulmonary branches of the vagus giving rise to cough may itself be secondary to some other condition; for example, measles, typhoid fever, or some other infectious disease; to chronic heart disease or to Bright's disease.

Diseases of the Heart.—Pericarditis is sometimes attended by a hard painful cough. Valvular disease, and especially mitral stenosis and mitral incompetence during the stage of failing compensation, are accompanied by cough, often with watery or blood-stained expectoration.

Aneurysm of the aorta may give rise to very distressing cough, either by direct pressure on trachea or bronchus, or by irritation of the recurrent laryngeal nerve.

Any condition in which the heart's action is feeble, and where consequently the circulation through the lung is carried on imperfectly, may give rise to a troublesome cough.

Diseases of the Abdominal Viscera.—The "stomach cough" associated with indigestion has already been mentioned. The existence of a true "stomach cough" has been doubted, and it may be admitted that such a diagnosis may legitimately be regarded with some scepticism. In many cases of the kind the cough appears to be due to pharyngeal irritation associated with the dyspepsia. The irritation produced by entozoa is said occasionally to give rise to cough. Cough may be present in pathological conditions of the ovaries and tubes, and pregnancy is also regarded as an occasional cause.

Nervous Cough.—The term "nervous cough" may be applied to all forms of cough in which no cause of local irritation can be discovered. Nervous cough may be a mere habit, more annoying to the hearer than to the subject. Its occurrence is often associated with emotion, particularly the emotion of speaking in public. Some forms of nervous cough have already been referred to.

Hysterical cough is comparatively common in cases of hysteria. It may be very severe and persistent, and give rise to serious apprehensions as to the condition of the lungs.

The *periodical night cough* occurs in children. The cough comes on in paroxysms about the middle of the night, and may last for several hours. It is probably often due to irritation of the vagus by enlarged lymph nodes.

About the time of *puberty* a loud barking cough, recurring at frequent intervals, is not uncommon in boys of neurotic constitution.

Diagnosis.—Some forms of cough, such as the cough of pertussis or of aneurysm, are very characteristic, and many more are easily traced to their origin. The difficulty in diagnosis is greatest where no obvious disease of the pharynx, larynx, trachea, bronchi, or lungs can be made out, nor of the heart or aorta. Such cases should not be too quickly set down as nervous. Repeated examination should be made of all possible sites of local irritation. In children the ears and the naso-pharynx should be carefully examined, and the possibility of enlargement of glands of the tracheo-bronchial group must be borne in mind. The family history should also be inquired into, especially with reference to any tendency to tuberculosis or neurosis. Hysterical cough may be present in either children or adults. In women the possibility of pregnancy or of pelvic disease being responsible must be considered. In adults, and especially in elderly patients, any evidence of feebleness of the circulation, such as weakness of the pulse or coldness of the extremities, is of importance.

Treatment.—The treatment of cough due to diseases of the respiratory organs or of the heart and aorta will be found detailed under the appropriate headings. Excluding these, the throat will frequently be found to be the seat of irritation, and pharyngitis, enlarged tonsils, or elongated uvula will require local treatment. Gargles, sprays, pigments, pastilles, are all useful in suitable cases. The great benefit often to be derived from constitutional treatment, sea-bathing, and an outdoor life is not always sufficiently recognised.

Inhalations are of value when the irritation proceeds from the larynx, and the use of the bronchitis kettle and copious warm alkaline drinks are most valuable adjuvants in all conditions where the cough is aggravated by scantiness or viscidity of the expectoration.

"Ear" cough usually disappears immediately on the removal of the cause of the local irritation, often a plug of wax or a foreign body, such as a bead, inserted in the ear by the patient.

"Stomach" cough is to be treated by a suitable dietary, and the use of gastric sedatives such as bismuth and hydrocyanic acid.

The barking cough of puberty is best treated by a plain but sufficient dietary, active out-door exercise, early hours, and the use of tonics. Sir Andrew Clark was in the habit of ordering the syrup of the bromide of iron, with small doses of arsenic.

The danger attending the indiscriminate and injudicious use of sedatives and narcotics (especially opiates) in the treatment of cough, and the disastrous consequences which may result in young children and elderly patients especially, are pretty generally recognised. Nevertheless, it must not be forgotten that severe and prolonged coughing, coughing out of all proportion to the amount of the expectoration, if unrestrained, involves a great strain on the lung, and may result in serious mischief to the lung tissue. Where a sedative is required small doses of chloral or of the bromides are often preferable to opiates. Children are not infrequently kept awake by an irritative cough which can be at once relieved by a few grains of chloral.

The bromides are of value in all forms of paroxysmal cough, and ammonium bromide has long been the favourite remedy in pertussis. Some cases of pertussis are greatly relieved by antipyrin in doses of about 1 grain for each year of the child's age.

In elderly patients a chronic cough associated with slight bronchial catarrh or congestion of the bases, and which has failed to benefit by the usual remedies, will often disappear under a course of digitalis in small doses. Strychnine is also of value in some of these cases.

Cranial Nerves.

THESE will, for convenience, be considered as follows:—

GROUP I.
- *a.* Olfactory. See Nose.
- *b.* Optic. See Optic Nerve and Retina.
- *c.* Third, fourth, and sixth. See Ocular Muscles.

GROUP II.
- *a.* Auditory.
- *b.* Facial.
- *c.* Fifth.
- *d.* Glossopharyngeal.
- *e.* Hypoglossal.
- *f.* Spinal Accessory.
- *g.* Vagus.

Craniotabes. *See* Rickets.

Cretinism.

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Definition.—Cretinism (congenital myxœdema) is the peculiar condition of arrested bodily and mental growth and development, with increasing deformity and debility, which results from a congenital deficiency of thyroid secretion due either to absence of the gland or to some morbid change interfering with its function.

The name is also generally, and perhaps conveniently, applied to the cases where defective action of the thyroid begins in early childhood (juvenile myxœdema).

Causation.—That the essential cause of cretinism is deficiency of the thyroid secretion is now an accepted fact. The morbid influences, however, which lead to disease or atrophy of the thyroid gland, and thus abolish its secretion, are still to a large extent unknown. They are probably different in the endemic and sporadic forms of the disease.

Endemic cretinism is generally met with in mountainous districts. It is always associated in distribution with endemic goitre, and a large proportion of deaf and dumb children are found in the same districts and families. The cause of these three conditions is evidently the same, and is known to be present in the drinking water. It is, in all probability, a micro-organism (Kocher), although this has not yet been absolutely proved.

All inquiries into the primary cause of *sporadic cretinism* have hitherto had only negative results. The disease very often occurs in large and otherwise healthy families. Two or more cases are, however, sometimes found in one family, and it is also not very uncommon for cases of infantilism, achondroplasia, and other forms of dwarfing to be present among the brothers and sisters.

In cases of *juvenile myxœdema* there is often a history of one of the infectious diseases having occurred shortly before the symptoms were noticed.

Distribution.—Endemic cretinism is almost unknown in Great Britain, but a few cases are to be met with, especially in Derbyshire, Somersetshire, Yorkshire, and Westmoreland. It occurs to a considerable extent in the mountainous regions of Europe, especially in Switzerland, France, and Italy, and has been reported as prevalent in various parts of North and South America, China, India, Madagascar, etc.

Sporadic cretinism is a comparatively rare disease, but cases of it have been reported from nearly every quarter of the world. There seems no reason to connect its occurrence with the physical characters of the district in which it is found.

Description.—*Infancy.*—Even in severe cases of cretinism there seems to be very little wrong with the child at birth. If the mother is observant, however, she soon notices that the baby is backward and apathetic—crying and laughing very little, and that the bowels are obstinately constipated, also often that the tongue seems too large.

On examining the infant cretin we are struck by his puffy, expressionless face and wrinkled forehead (Fig. 1), and on measurement he is found to be undergrown, although as yet there is no noticeable disproportion between the trunk, head, and limbs. His hands are characteristically broad and short. The temperature is subnormal. The thyroid is generally absent in sporadic cases, while in the endemic form there is usually a goitre.

Childhood and Youth.—As the child grows older his development lags behind, and the characteristic cretinous appearance becomes steadily more striking. The myxœdematous swelling increases in the face and elsewhere, the belly becomes more prominent, and marked lordosis develops. There is generally an umbilical hernia. Circumscribed fatty swellings appear above the clavicles and in front of the axillæ. The dryness of the skin increases, and the hair is very scanty and dry. The fontanelle remains widely open. The milk-teeth may or may not be delayed in appearance, but generally they remain too long in the gum, and they may all be present even as late as the eighteenth year. The child's growth and activity are greatly interfered with, so that at ten or twelve years old he often has the size and the feebleness of a boy of three or four. The mental condition in an ordinarily severe case of the disease is that of imbecility, but in slighter forms of the disease the child seems merely backward. He is dull and apathetic, slow of movement and of apprehension, but neat and tidy in his ways, and docile and quiet if not teased. Speech is generally long of being acquired, and the words used are few.

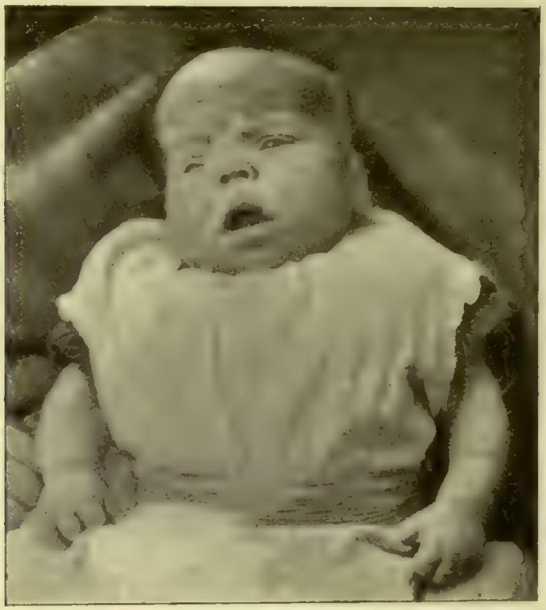

FIG. 1.

Adult Age.—In spite of their debility, cretins not infrequently live to forty, fifty, or even sixty years old. The adult cretin shows the disease in its most marked form (Fig. 2). He is generally about three feet in height, with large brachycephalic head, thick dry redundant skin, scanty coarse hair, and an open fontanelle. His trunk is stunted, and he has marked lordosis and a prominent belly; there is often also lateral curvature. The limbs are short and thick-set, with prominent hard muscles. The gait is very feeble and waddling. The extremities are always cold. There is little or no pubic or axillary hair. In the male the external genitals are like those of a child, while in the female the breasts remain quite undeveloped.

Menstruation is very late of appearing and very irregular, if it comes at all. In the few cases in which pregnancy and delivery have taken place, the infant, although not myxœdematous, has been atrophied or hydrocephalic at birth, and has died soon after. The cretin's mental condition does not improve as age advances. Although very dull, he understands and observes more than he appears to, but he has little or no initiative. His interests are exceedingly limited. He is shy, morbid, and solitary.

During recent years a number of slighter cases have been described by Hertoghe and others as really instances of cretinism, although they lack many of the usual symptoms. In these there is little beyond stunted growth, delayed closure of the fontanelle, and some degree of mental dulness. The confirmation of the diagnosis is to be sought in the marked improvement which follows thyroid treatment.

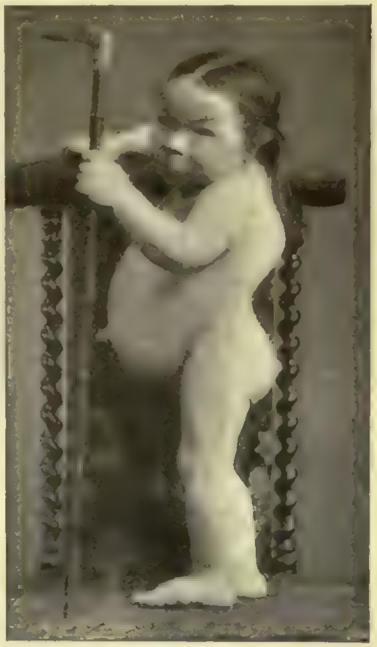
Fig. 2.

Diagnosis.—The two morbid conditions most apt to be mistaken for cretinism are achondroplasia and the so-called "Mongolian" type of imbecility.

The resemblance which achondroplasic dwarfs have to cretins at birth is in some ways striking, but it is *adult* cretins they resemble. The extreme shortness of limbs which they exhibit is not a characteristic of cretin infants, while their soft natural skin and hair, their normal temperature and mental condition, and the peculiar formation of their hands (see "Achondroplasia") enable them to be readily distinguished.

"Mongolian" imbeciles resemble cretins in their backwardness, their frequently protruding tongue, scanty dry hair, and dwarfed stature. They differ in being less stunted in growth, in their physiognomy, in the shape of their hands, and in their general appearance and mental characteristics (see "Idiocy and Imbecility.")

Morbid Anatomy.—In the endemic form of the disease, goitre is present in about 75 per cent of the cases, while in the remainder the thyroid is absent.

In most cases of sporadic cretinism no trace of a thyroid can be discovered; in a few the gland is atrophied, and shows cirrhotic changes; rarely there is a cystic goitre.

Treatment.—The treatment of cretinism, like that of adult myxœdema, consists in the continued administration of some preparation of thyroid gland by the mouth. The thyroid of the sheep is that generally used, and it may be given raw or in such form as the Thyroideum Siccum or Liq. Thyroidei (B.P.) or various other dry preparations. One of the most reliable of these, and one which seldom disagrees, consists of the dried colloid matter of the thyroid (Hutchison). The dose must vary according to the age, size, and strength of the patient, and according to the intervals at which the remedy is administered.

The fresh raw thyroid is probably more active and trustworthy than any of its preparations. It may be given to a young child, to begin with, in doses of $\frac{1}{16}$ to $\frac{1}{8}$ of a gland *twice a week;* or in double these doses to an older child or adult. After some time one whole lobe may often be taken each time with advantage. It is a curious fact that the improvement in cases where thyroid is given twice a week is just as continuous and satisfactory as that seen where the remedy is administered in small doses daily or every few hours.

Generally, however, it is more convenient to use one of the manufactured preparations, and to give it daily. In a young infant we may begin with gr. i. of the dried gland, m. ii. of the Liq. Thyroidei, or gr. $\frac{1}{5}$ of the dried colloid matter. If these doses cause no unpleasant symptoms they may soon be increased. In adolescent or adult patients, from three to five times these doses may be given daily to begin with.

During treatment the patient may go about as usual, and no special diet is required. The temperature, weight, growth and general health must be closely watched. Should the patient become feverish, and show signs of sickness, headache, etc., this indicates rest in bed and a diminution of the dose. Often after some weeks or months an increase of the dose is necessary to maintain the improvement satisfactorily.

In treating adolescent cretins it is very important to prevent, if possible, the bending of the legs which is apt to follow their greatly increased activity. For this purpose they should be made to lie as much as possible.

THE RESULT OF TREATMENT.—When the treatment is carefully carried out rapid and continuous improvement results. The temperature rises to and remains at the normal level. The unnatural swelling quickly disappears from the face and other parts of the body. The features lose their unnatural thickness and become more mobile, and the eyes look much brighter. At the same time the tongue ceases to be protruded, the voice becomes less guttural, and the child no longer snores at night. The abdomen diminishes greatly in circumference, and if an umbilical hernia is present it disappears. The fatty swellings also vanish at an early stage of the treatment.

The skin loses its harsh and dry feeling and becomes soft, and the cheeks show a natural flush. In young patients the hair falls out, at first, in considerable quantities, but is soon replaced by a new crop which grows more rapidly and is softer and often of a different shade of colour.

At first there is a considerable loss of weight, with a relaxed condition of the muscles and ligaments, but in the course of six months, if not before, the patient gains flesh considerably. The limbs also become firm and strong, and the back straighter and more shapely. The retarded evolution of the teeth is actively resumed.

The growth of the skeleton is perhaps the most striking change of all. It begins at once and proceeds rapidly. The patient often gains 2 in. in height within the first two months, and may make as much as 6 or 8 in. in the first year. After that the rate of growth diminishes and approximates to the normal. The appetite is greatly increased by the treatment, and the bowels generally become regular in action. The mental improvement which occurs is apt, at first, to be greatly overestimated by the parents, because the child *looks* so much brighter and his movements are so much livelier than before. Within six months, however, there is unmistakable advance, and this continues and increases—the better nourished brain becoming increasingly capable of work. The children become more inquisitive, more independent and enterprising, and more inclined to do things. They lose their shy, morose, self-centred disposition, and become bright, happy, childlike, and sociable.

In the milder cases, the arrears of bodily growth are soon fully made up, and the state of the intellect approaches, if it never quite reaches, the normal. In a certain proportion of cases, however, while the bodily recovery is more or less complete, the child remains an imbecile.

In adolescent cases (from sixteen to twenty-five years) the improvement is very great, but a considerable degree of deformity remains, owing to the lower limbs not growing in proportion to the rest of the body. There is also, at this age, a strong tendency for the legs to become much bowed, and this is extremely difficult to prevent.

In adults (after about thirty) the effect of treatment is much less. In them there is usually a growth of one or two inches during the first few months, and none after. The appearance of the face improves greatly, menstruation becomes more or less regular and the breasts enlarge. Even at this age, however, the treatment is advantageous because of the great improvement which it causes in the patient's general health, and especially in his happiness. Although he still remains a child in mind and a dwarf in body, he begins for the first time to take an active pleasure in seeing and doing things and in associating with other people.

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Criminal Responsibility.—In holding a person responsible (*i.e.* liable to punishment) for his misdeeds the law rests on two assumptions or postulates regarding human beings. These are (1) that the individual can distinguish between "right" and "wrong" in the concrete case; and (2) that he is possessed of will-power adequate to control his impulses, and to control them in the light of that knowledge of right and wrong. The law *presumes* these capacities to be present—presumes a man to be *sane* according to this standard. Where, however, it can be established that, owing to mental infirmity, these capacities, or either of them, are lacking in any individual, the general rule of responsibility is relaxed. If a criminal act be unmistakably the result of *insanity* in the perpetrator, the accused is held to be not responsible therefor.

The decision of the question whether an accused person is, on the ground of insanity, to be exempted from responsibility for an act with which he is charged, is, of course, the function of the jury, under direction of the judge. But it is the duty of the medical expert to assist them in coming to a right verdict in the matter. It is the purpose of the present article merely to indicate, for the guidance of medical witnesses, the standpoint from which the matter will be investigated judicially by setting forth the generally accepted criteria of *legal* insanity. This article does not attempt to follow the controversy between medical and legal authorities as to what degree or what forms of mental derangement ought to exempt from responsibility for crime.

The authoritative statement of the law of England bearing on criminal responsibility is to be found in the answers of the supreme judges to the questions submitted to them by the House of Lords in 1843 (commonly known as "The Rules in MacNaughton's Case," 4 *St. Tr.* N. S. 847). The leading feature in these rules, as compared with the views previously enunciated by legal authorities in England, was the repudiation of a knowledge of right and wrong merely *in the abstract* as any test of sanity, and the substitution of the question whether the accused at the time of the act was labouring under such a defect of reason from disease of the mind as not to know the nature and quality of the *particular act* he was doing. Baron Hume (in his Scottish treatise on *Crimes*, i. p. 37) had put the matter thus: "The question must be relative to the particular act done, and the accused's knowledge of the situation in which he did it. Did he as at that moment understand the evil of what he did? Was he impressed with the consciousness of guilt and fear of punishment?" The English judges followed the Scottish authorities by laying it down that the inquiry must be directed to the accused's mental state in relation to the particular act done.

To establish a defence on the ground of insanity, therefore, it must be clearly proved that at the time of committing the act the accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong. "If the accused was conscious that the act was one which he ought not to do, and if the act was at the same time contrary to the law of the land, he is punishable."

If a person under an insane delusion as to existing facts commits a criminal offence in consequence thereof, he is not necessarily freed from responsibility on the ground of insanity. Much depends on the nature of the delusion. The accused's responsibility is to be determined by considering whether, assuming as real the facts with regard to which the delusion exists, there would or would not be legal justification for his conduct. Thus, if under the influence of his delusion, he supposes another man to be in the act of attempting to take away his life, and he kills that man, as he supposes, in self-defence, he would be exempt from punishment. If, on the other hand, his delusion was that a man had only injured him in reputation or fortune, and he killed him in revenge for such supposed injury, he would be liable to punishment. This statement of the law given by the English judges has not commended itself to medical men. As a test of responsibility it appears to be vitiated (as Dr. Maudsley points out) by the assumption that a man, having an insane delusion, has the power to think and act in regard to it *reasonably;* that at the time of the offence he ought to have and to exercise the knowledge and self-control which a sane man would, were the facts with respect to which the delusion exists real. It, however, stands as authoritative in the law of England; but, fortunately, it has not been very logically applied in actual cases. Its corrective is probably supplied by the more general rule of reference in each case to the accused's knowledge of right and wrong in regard to the particular act. In Scotland, on the other hand, it has been judicially recognised that a man may be entirely insane, and yet may know well enough that an act which he is doing is forbidden by the law of the land.

If a man have not a sane mind to apply his knowledge, the mere intellectual apprehension of an injunction or prohibition may stimulate his mind to do an act simply because it is forbidden, or not to do it because it is enjoined (Lord Moncreiff in case of Miller, 1874, 3 Couper's *Reports*, 16).

The "Rules in MacNaughton's Case" do not deal with the subject of *moral insanity*. For long the Courts rigidly refused to recognise such form of mental derangement. But *moral insanity* (meaning by that term mental derangement in which disorder of the moral faculties is more pronounced than disorder of the intellectual) is now undoubtedly accepted in the British Courts as freeing from responsibility or mitigating punishment according to the circumstances of the case. Further, *weakness of mind* of a lesser degree, not such as to free entirely from responsibility, is held either to warrant leniency in punishment, or, it may be, to justify a verdict of something less than the full crime charged, *e.g.* of manslaughter or culpable homicide instead of murder.

If insanity be proved, the question of responsibility is not affected by a consideration of what was the *cause* of the disorder. The fact that it was occasioned by accused's own acts (drunkenness or other excess) is not of consequence. Intoxication does not free from responsibility; insanity brought on by drunkenness will. Where a person killed another under a "momentary hallucination induced by drunkenness," it was laid down in a Scottish case that the jury might acquit of murder and convict of the minor crime of culpable homicide (Robertson or Brown, 1886, 1 White's *Reports*, 93).

Presently existing insanity may bar criminal proceedings, before, at, or after trial. In such a case it is, of course, the *present* mental condition of the accused that the medical witness must speak to; and therefore an examination of accused ought to be made as shortly as practicable before the inquiry—on the same day, if possible. In Scotland, where insanity is pleaded in bar of trial, the facts to be established (before a judge without a jury) are that the accused is presently insane, and incapable of giving intelligent instructions for his defence.

LITERATURE.—1. See STEPHEN'S *Hist. Crim. Law*, ii. p. 150; *Digest Crim. Law*, p. 173.—2. HUME on *Crimes*, i. p. 37.—3. ALISON. *Crim. Law*, i. p. 645.—4. HADFIELD'S Case, 1800, 27 *How. St. Tr.* 1281.—5. RAE'S *Med. Juris.* p. 159.—6. TAYLOR'S *Med. Juris.* ii. p. 557.—7. HAKE TUKE'S *Dict. Psych. Med.* i. p. 301.—8. MAUDSLEY. *Reponsibility in Mental Disease*, p. 88.—9. WOOD RENTON on *Lunacy*, pp. 885 *et seq.* *Monomanie Sans Délire*, etc.

Croup. *See* DIPHTHERIA, LARYNGITIS.

Crystalline Lens. *See* LENS.

Curettage, Uterine.

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IT is now fifty years since Récamier advised the scraping of the inside of the uterus with a sort of scoop with sub-acute edges in cases of metrorrhagia from "intrauterine fungosities"; but discouraging and even fatal results followed this first use of the curette, and it was not till 1865 that Marion Sims ventured to characterise the operation as a practice "now recognised as legitimate." Sims' curette was a sharp one, and so was the spoon curette of Simon introduced in 1872; but the blunt instrument invented by Thomas, and popularised in this country by Mundé, by its almost complete safety did a great deal to establish the curette in the favour of the profession. Its chief use was as a means of diagnosis. Since 1878 the sphere of usefulness of the uterine curette has been enormously widened; sharp and blunt and flushing instruments are employed, and the operation of curettage is looked upon as one requiring care and skill, and not simply as a trifling diagnostic method which could be safely carried out on the consulting-room couch.

The INDICATIONS for curettage may be divided into therapeutic and diagnostic; and of these the former are nowadays regarded as the more important, and will be considered first.

Therapeutic Uses.—(1) *Hæmorrhage* from the genital organs is one of the commonest and most fully established uses of the curette. The bleeding may be from the cervix uteri, or even from the vaginal walls, as in cases of malignant disease of these parts, and then the curette acts only as a palliative; in the great majority of cases, however, the hæmorrhage is from the body of the uterus, and curettage is at any rate expected to be curative. Uterine corporeal hæmorrhage may be due to retained products of conception, as after an incomplete abortion; then the cavity may be cleared out with the index finger, which is, under the circumstances, the best curette; but if the cervical canal has closed and the bleeding continues, notwithstanding the lapse of some days or weeks, then dilatation and a thorough curetting of the interior will be needed, for decidual remains are often very firmly fixed, and need not be of much size in order to set up a great amount of hæmorrhage. Again, the hæmorrhage may be due to chronic non-septic hyperplastic endometritis, and in such cases the removal of the greatly thickened endometrium by the curette (blunt or sharp) with previous dilatation of the cervix will often give immediately satisfactory results, especially if the curettage be followed by a thorough cauterisation of the uterine interior. It cannot be said that hæmorrhage from a uterus affected with fibroid tumours has been more than temporarily checked by curettage, for the mucous membrane under these circumstances is not always hypertrophic (it may indeed be atrophic), and the congestion may be seated in the muscular coat, which is of course beyond the reach of the scraping. In the case of sarcomata and carcinomata of the body of the uterus the curette is only a palliative means of treatment, but it has a value as a diagnostic, and, further, it may be used as a preliminary procedure before the performance of vaginal hysterectomy for these uterine tumours.

(2) *Septic and other infected states* of the uterus, and even of the Fallopian tubes, have during recent years been treated by curettage; but all gynecologists are not agreed as to the value of this indication, and, of course, all infected states are not equally amenable to curettage. In acute septic endometritis, for instance, following abortion, or even labour at the full term, the curette must be used only after consideration, and not as a routine plan of treatment; but even under these conditions it has its uses, especially if intrauterine douching, iodoform packing, and the antistreptococcic serum fail to reduce the temperature. Great care must be taken not to perforate the soft walls of the puerperal or post-abortum uterus. In chronic infected states in which the uterus is enlarged and displaced, menstruation disordered, and purulent and profuse leucorrhœa in existence, the curette often gives the best results. Even in cases in which the appendages are infected it has been recommended that the uterus be curetted in order to drain pus-containing tubes through it.

(3) *Dysmenorrhœa and sterility* form yet other indications for curettage, but it is often difficult to separate these from the chronic infected states.

At any rate it is probable that the straightening of the uterus and the dilatation of the cervical canal may be as efficacious as the curettage in the improvement which undoubtedly sometimes follows. (4) In the *induction of abortion* for dangerous conditions in pregnancy, such as incoercible vomiting, the curette (preferably the blunt variety) may be employed, especially when it is important to carry out the induction rapidly and with small loss of blood. (5) Curettage may be used as a *prophylactic and preliminary procedure* when it is intended to operate on the vaginal walls or cervix, or to do hysterectomy, in order to prevent the infection of the wounded surfaces with septic material, etc., from the uterus.

The diagnostic uses of the curette consist in the removal by this means of scrapings of uterine mucous membrane and their examination under the microscope in order to separate conditions of simple endometritis from sarcoma and carcinoma, and even from fibro-myoma of the uterus.

The Technique.—When the condition calling for curettage permits the fixing of dates beforehand, the time chosen should be the week after the menstrual period. On the day before operation the patient should keep in bed, her urine should be tested, and some opening medicine should be given, to be followed by an enema next morning. The vulva and vagina should be thoroughly scrubbed out, and the operator and assistant should take the same personal precautions to ensure surgical cleanliness as if a laparotomy were anticipated. The armamentarium consists of a curette, which may be either Martin's sharp curette (which closely resembles that recommended by Récamier), or an instrument with a hollow stem, by means of which a stream of antiseptic solution can be sent through the uterus (flushing curette); a vaginal speculum, such as Sims'; two or more strong-toothed volsellæ with clips; a set of cervical dilators, such as Hegar's; three or four uterine sounds armed with cotton wool; a male catheter, No. 10; an intra-uterine douch and a pair of curved packing forceps; some pledgets of gauze, and iodoform gauze in strips for packing. There should be at hand a bottle of iodised phenol for applying to the interior of the uterus on the armed sounds, if regarded necessary; there must of course be an anæsthetic (chloroform), and an antiseptic solution (perchloride of mercury, 1 in 5000). The patient, having been anæsthetised, should be placed in the lithotomy position on a table in a good light. The field of operation should be protected with sterilised towels, and the hips should be raised upon a pad of antiseptic cotton covered by mackintosh. The external genitals and vulva should be again thoroughly washed and scrubbed, and the urine drawn off. The speculum is then passed and held in position exposing cervix and vaginal vault, and the operator seizes and drags down the cervix by means of the volsellæ, one being attached to the anterior and the other to the posterior lip. If the uterus be fixed by pelvic adhesions this should not be attempted. The dilatation of the cervical canal is now begun, unless it is already sufficiently open to allow the passing of the curette; the Hegar dilators, which have been carefully sterilised, are now slowly introduced one after another, beginning with perhaps a No. 3 and working up to a No. 13 or 14, and always allowing a minute or two to elapse between successive ones. From fifteen to twenty or even thirty minutes should be allowed for this part of the operation. Each dilator should be warmed and oiled before introduction. The curette is now passed into the uterus and the walls systematically scraped with it (first anterior, then posterior, and so on), the scraping, which is accompanied by a creaking ("le cri uterin"), being controlled by the left hand placed over the abdomen. The contents of the curette should be floated off into a cup of clean water for future microscopic examination. Next, the uterine cavity may be washed out with an antiseptic solution, and iodised phenol applied to the denuded endometrium on armed sounds if the caustic effect be desired. Finally, the uterus and vagina should be packed with a long strip of iodoform gauze, a pad of antiseptic absorbent wool should be placed over the vulva, and the whole held in position with a bandage. The patient is then put back to bed and kept quiet. The urine may require to be drawn off, but it is often passed naturally. If the curettage has been done for suppurative conditions, the packing may need to be frequently changed; if not, it may be allowed to remain in for two or three days. The patient should be kept in bed for five days or a week, for longer if the operation has been performed for grave conditions; she ought to rest in bed at the next menstrual period, as the discharge is sometimes excessive then, and marital relations should not be resumed for eight weeks in order to permit of the complete restoration of the endometrium. It has to be borne in mind that curettage does not prevent future conception; it has indeed cured sterility.

The DANGERS of uterine curettage, if carried out with surgical cleanliness, are not many or great. Occasionally perforation of the uterine wall has occurred, especially when the operation has been done in the puerperium; then if grave symptoms, such as hæmorrhage or prolapse of intestine, appear, it may be necessary to perform hysterectomy, but in many cases no harm has resulted. Sepsis is not a common danger nowadays when curettage is looked upon as an operation and not as a diagnostic method to be done in the consulting-room or with the patient in bed. Abortion may result through want of diagnosis of pregnancy, therefore it is always well to inquire into the menstrual history, and to make a careful bimanual examination before beginning the cervical dilatation. Complete obliteration of the uterine cavity has in a few instances followed curettage, possibly on account of a sort of superinvolution. Rupture of purulent collections in the pelvis is a danger which used to be much feared, and tubal disease and perimetritis were formerly regarded as CONTRA-INDICATIONS to curettage; but the risk has probably been exaggerated, and it is now held by some gynecologists to be good treatment to use the curette in these very conditions which were regarded as contra-indications. Of course under these circumstances the alternatives are vaginal hysterectomy or laparotomy. Pregnancy is a certain contra-indication, save when it is desired for some good reason to induce abortion.

Cut Throat. *See* NECK.

Cystinuria. *See* URINE.

Cystitis. *See* BLADDER.

Cystoscope, the.—To examine the interior of the bladder visually without a cutting operation is no new idea. In 1805 Bozzini of Frankfurt devised and exhibited an instrument for this purpose, which, however, was found to be quite inadequate because of insufficient illumination. Desmoreau, in 1854, introduced a cystoscope which, in 1865, was further elaborated by Dr Cruise of Dublin. These and all other instruments prior to 1877 may be considered the first stage of the cystoscope, as the light for illumination was *external* to the bladder, but in that year Dr Max Nitze of Berlin introduced the platinum loop instrument with the light internal, which, although it had various objections, and was soon condemned because of its cumbrous size and defective light, initiated the second stage of the cystoscope or light *internal* idea, and led up to the more perfect instrument of to-day. Not until 1887, however, did surgeons find an instrument worthy of a place in their armamentarium, when, for the first time, the "incandescent lamp" cystoscope was introduced. This instrument has been variously modified, but the Leiter cystoscope, after the model of Mr Hurry Fenwick, serves excellently for thorough examination of the bladder.

The Instrument.—Two forms of the cystoscope—the *anterior* and *posterior*—are in use, but in practice the former is alone necessary. It resembles a sound in shape, and consists of the beak, the shaft, and the ocular position. The beak is hollow, contains the lamp used for illumination, and has a window through which the light is emitted. From the lamp two insulated wires pass along the interior of the shaft to the ocular portion, where they are connected with the electrodes of a battery, the source of electro-motive force for light. The shaft is hollow, and contains a telescope. At the junction of the shaft and beak, in line with the window in the beak, there is a prism which refracts the rays of light from the object illuminated on to the end of the telescope, and therefore into line with the observer's eye, so that the object is brought into view. On the rim of the ocular portion, upon the same aspect as the window and prism, there is a knob which indicates their position, so that as the instrument is moved to and fro, or turned round within the bladder, we can determine the part of the bladder wall which we examine. When using the instrument it is focussed by withdrawing it from, or approaching it to, the object under examination until the best definition is got. It is necessary to remember that the image is inverted. With the anterior cystoscope the whole surface of the bladder can be seen, but naturally the most difficult portion to bring into view is that immediately around the urethral opening. A suitable battery, provided with a rheostat, is that figured in Schall's catalogue No. 1192.

To use the Cystoscope.—The most careful aseptic precautions are required just as in the use of other bladder instrumentation. The cystoscope cannot be boiled without injury, and must therefore be purified by steeping it for a time in carbolic acid or lysol lotion. The other usual precautionary measures to avoid rigors must also be adopted. The pain during examination is no greater than that caused by sounding, and it is only in exceptional cases that anæsthesia is required, but an anæsthetic may be exhibited in nervous patients or in those with sensitive bladders. In the female, from motives of delicacy, an anæsthetic may be necessary. In addition to these general measures there are three essentials for examination :—

1. The urethra must be of sufficient calibre to easily admit the instrument.
2. The bladder must be capable of holding at least six ounces of fluid.
3. The fluid in the bladder must be transparent.

Method of Examination.—The urine is drawn off with, by preference, a red rubber catheter. If it be clear—no blood or pus—from 6 to 12 ounces of warm aseptic solution are injected, but if need be the bladder is first washed out that the injected fluid may remain transparent. This should be done as gently as possible to avoid hæmorrhage, which might rapidly cause the fluid to be coloured, so that the examination would be difficult, although it is very rarely impossible from this cause. The cystoscope is now introduced, the circuit being *open*, and care taken that the beak is completely within the bladder before it is closed. The circuit having been *closed* the observer's eye is applied to the ocular portion, and an endeavour made to detect an air bubble, which is almost certainly present at the highest point of the fluid. This is seen as a shimmering globule, and indicates the position of the instrument—gives, so to speak, a landmark from which we can proceed methodically to examine the whole bladder wall. The normal bladder wall has a pale, pinkish white colour, with small but distinct blood-vessels ramifying on the surface. The pallor varies in degree proportionately to the distension. Bladders vary in their appearance, and it is only after several have been examined that the observer can say whether a departure from the usual appearance is consistent with health or not. During the examination the beak of the instrument must not rest continuously on the wall, as it may become hot and do harm ; but this is not apt to occur if ordinary care be taken. If an abnormality be seen it must be closely examined to determine its exact significance. A projection is more readily seen than a superficial lesion. Different meridians are examined by advancing and withdrawing the instrument, but in particular the margin of the *urethral opening; neck of bladder;* and *trigone* are examined. The neck of the bladder is congested in appearance and prominent, but normally smooth in its outlines. The ureteral openings are at each end of the base of the trigone, with it may be an inter-ureteral bar. They may in some instances be readily seen, but in others require to be closely looked for by judging the distance and proper angle from the urethral orifice. The openings are normally transverse or oblique slits, and may not be detected until the margins are separated by urine coming through them. It is necessary, therefore, to steadily watch for a minute or two the area where we expect to find the opening, so that as the urine is ejected the orifice is seen. If the urine be mixed with blood or pus from the kidney its ejection is more readily observed. Bright blood is readily seen (*v.* Case 1. *infra*). If the prostate be much enlarged the beak of the cystoscope must be considerably depressed to bring the trigone into view. When the examination is completed the circuit is opened and the instrument withdrawn. It is important to introduce and withdraw the cystoscope with the circuit *open,* as otherwise the beak, not being surrounded by water, becomes hot, and may burn the urethra. The instrument should not be kept for any time in the air with the circuit closed, as by fusion of the connections it may be injured.

Value of Cystoscopy.—In obscure genito-urinary conditions, both as a diagnostic and prognostic agent, the cystoscope is valuable. It may enable the surgeon to say definitely that the bladder is or is not the source of hæmorrhage, the origin of which cannot be otherwise determined. That is equivalent to deciding in some cases whether the bladder is to be opened or a nephrotomy performed. Further, even if the bladder be the seat of a tumour cystoscopy may show whether it is suitable or not for removal. Wherever there is doubt as regards the site or nature of disease causing hæmaturia or pyuria, or when other means have failed to give an accurate diagnosis, cystoscopy should be used. There are two considerations, however, of much importance, which should invariably be borne in mind:—*First,* cystoscopy, just as all instrumentation, should as a rule be avoided in vesical tuberculous disease; and, *second,* in patients who are seriously ill, or in whom instrumentation is apt to be followed by aggravation of symptoms, whatever the reason, operation if needed should immediately follow the examination. In those cases of vesical tuberculosis, where the bladder is much inflamed or contracted, cystoscopy should never be resorted to, as in these harm is frequently done; simply it may be by over-distension of an inflamed bladder; and the method is brought into disrepute. When, however, there is no obvious tuberculous disease of the prostate, and the bladder is of good capacity, if there be doubt regarding the source of tubercle bacilli or pus, cystoscopy is valuable to determine whether the kidney is diseased and the bladder healthy. Nephrectomy in primary tuberculous disease of the kidney may be curative, and the decision to perform the operation may depend upon cystoscopic examination showing absence of disease in the bladder. This aspect of the subject can best be appreciated by the consideration of illustrative cases, of which the following may be considered fairly typical:—

Case 1.—Mr. J. D., æt. 47, complained of pain in the left side and blood in the urine. There was no other symptom, and examination discovered no disease in either the kidney or prostate. Cystoscopic examination showed blood coming from the left ureteral orifice. Nephrotomy was performed, but it was only upon cutting into the kidney that a tumour (sarcoma) was found and nephrectomy carried out. For one year the patient remained in good health, but then a swelling appeared at the seat of the incision, and death resulted one and a half years after operation.

Case 2.—M. S., male, æt. 60. Hæmaturia only symptom. Cystoscopy showed a pedunculated villous tumour on the right inferior aspect of the bladder about one inch above the right ureteral orifice. The tumour was removed by suprapubic cystotomy. The patient made a good recovery, and there was no sign of recurrence for three years. Now, four years from date of operation, the patient suffers from hæmaturia, and a large mass can be palpated in relation to the bladder.

Case 3.—Miss D., æt. 25 ; chief complaint, frequency of micturition. The urine contains a considerable quantity of pus. Pain in the right loin has been present off and on for four years, and on palpation the kidney is felt to be enlarged. Cystoscopic examination showed a normal bladder. No pus was seen coming from either ureteral orifice. Nephrectomy of the right kidney was carried out. The kidney contained large vomicæ filled more or less with caseous material.

Case 4.—J. S. (male), æt. 50, had symptoms of stone in the bladder, but sounding failed to detect one. Cystoscopy showed a small stone behind a freely movable pedunculated middle lobe of the prostate. An attempt to crush the stone failed as it could not be grasped, and suprapubic cystotomy was had recourse to. This case illustrates that, even when no deep post-prostatic pouch is present, a stone may be missed by the sound. That cystoscopy is of value in cases of post-prostatic pouch for the detection of calculi is undoubted, but the examination and operation should, if possible, be carried out at the same time. In such cases castration is contraindicated, as the stone or stones must be removed to get rid of the symptoms.

Many cases, differing from the above, might be quoted to emphasise the importance of cystoscopy, if space permitted, but those related will serve to show that assistance in diagnosis may be got by its use in (*a*) tumours, and (*b*) calculi of the bladder, and in different affections of the kidney. To detect the presence and nature of foreign bodies the instrument is also valuable. From the point of view of prognosis valuable aid is also got. Thus in tumour of the bladder, if a patient have little bleeding and no pain or frequency of micturition, but suffers from an irremovable tumour, cystotomy should be avoided as a rule. Rest to the bladder by cystotomy may diminish the rapidity of the growth of the tumour, but it may, on the other hand, permit an intrinsic growth to become extrinsic, so that eventually much greater discomfort and a more rapidly fatal result ensues. Cystoscopy, by showing the extent and attachments of the tumour, may prevent an operation which would be hurtful to the patient ; while, on the other hand, it may enable the surgeon more strongly to advocate operation, the diagnosis being certain and the hope of cure probable. The latter point is shown in case 2 quoted above, while the following case illustrates the former :—

Case 5.—Mr. R., æt. 58, three months ago, for the first time, had causeless hæmaturia which lasted for a few days. He had a second attack ten weeks later, and at the present time suffers from a third attack. There is no other symptoms, and external examination is negative. The urine contains many large multinucleated cells. Prostate not enlarged on examination per rectum. Cystoscopic examination showed a tumour attached to the prostate. From its extent and position I believed it to be inoperable, and advised no operation.

To decide whether a tumour is operable or not is far from easy, and in some cases impossible, but in other cases there is not much difficulty. If in doubt, I think the patient should have a cystotomy. The cystoscopic examination must be very thorough, and should preferably be carried out with different degrees of distension of the bladder. A tumour in an imperfectly distended bladder may appear as a mass, while if the bladder be more fully distended the growth may be seen to be made up of several separate masses affecting a much larger area of the viscus. I have seen this in two cases recently, in one of which cystotomy showed the disease was inoperable, while from cystoscopy I believed the disease could be eradicated.

The subject of cystoscopy has in the last ten years been much widened by the introduction of apparatus to treat conditions per urethram, or to catheterise the ureters. Both of these procedures I believe to be of little practical importance in the male, and sometimes, indeed, distinctly injurious. In the female, however, the method introduced by Dr. Kelly, of Baltimore, is more valuable, although short of ureteral catheterisation, equally good results can in the majority of cases be got by the anterior internal light cystoscope without the disadvantage of urethral dilatation incident to the use of Kelly's cystoscope. Examination by Dr. Kelly's instrument is simple, and ureteral catheterisation with it can be rapidly executed. A complete account of the instruments required for examination and the technique is contained in Kelly's work *Operative Gynecology*. The fundamental principles of examination are:—

1. The introduction of a simple cylindrical speculum.
2. The atmospheric distension of the bladder by posture.
3. The illumination and inspection of the bladder by a direct light.

The instruments required are: A strong light, a head mirror, vesical specula with obturators, a urethral calibrator and dilator, an evacuator for removing urine, and a ureteral searcher.

Local anæsthesia may suffice, and undue dilatation of the urethra is unnecessary.

LITERATURE.—1. Fenwick's *Electric Cystoscopy*.—2. Kelly's *Operative Gynecology*,

Dactylitis. *See* Hand, Injuries and Diseases of.

Deafmutism.

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By dumbness we mean the want of power to articulate sounds. All classifications of cases of dumbness are open to objection. The following two great divisions require special discussion:—

1. Deafmutism or deafdumbness. Dumbness due to deafness.
2. Dumbness associated with idiocy or dementia.

In addition to these, dumbness may be the result of damage to the brain, *e.g.* aphasia, where there is sometimes entire loss of speech, due to a lesion in the third left-frontal convolution; apoplexy, bleeding on or into the brain; embolism of a cerebral artery; tumour of brain, etc.

Further, dumbness may be part of a more general paralysis, such as lead paralysis, or it may be due to pressure on the hypoglossal nerve. Dumbness may also be a feature of hysteria.

Deafmutism.—The deaf and dumb are not otherwise physically peculiar. Placed under similar conditions they develop equally with hearing children. At six or seven, when the deaf child enters school, he is like the hearing child of two or three years, and it is a curious fact that his head is half an inch less in circumference than the hearing child of the same age. Such is the influence of hearing on the development of brain.

All deaf mutes are not stone deaf. Most hear loud noises or shouting, some distinguish vowels and consonants, and I have had them beat time accurately to music of all kinds when the sounds of the phonograph were conveyed to their ears by the tubes belonging to the instrument, although their eyes were covered carefully to prevent their seeing the manipulations about the instrument. But all deaf mutes are so deaf that they cannot hear ordinary talk and cannot be taught with hearing children. Speech is normally the result of hearing, and where there is little or no hearing the child will not speak. Further, if at four, five, or six years he lose his hearing, his speech will leave him.

Deafmutism is either congenital or acquired.—In Britain about 50 per cent of the cases belong to each of these two great classes. But in some parts of America and the continent of Europe, epidemic cerebral spinal meningitis raises the proportion of acquired cases, which with reference to the congenital cases bear a ratio of nearly two to one. Still, although epidemic meningitis is not common in Britain, diseases of the head, of which meningitis is the chief, are responsible for a large number; next to meningitis as causes of acquired deafness comes scarlet fever, and after this disease measles. These three diseases account for nearly 60 per cent of our cases of acquired deafness. Now, when these diseases take away hearing, they often take it all away, and, further, they often damage the intelligence of the child in other ways, hence come these two facts:—

1. When testing the hearing of the deaf and dumb it is common to find a congenitally deaf child with more hearing than a child who has lost his hearing after birth.
2. Teachers often find the congenitally deaf child bright and clever, and the acquired deaf mute slow and stupid.

Congenital deafness, as we have seen, accounts for about half the number of cases of deafmutism in Britain. Many cases supposed to be due to disease in early life are really cases of congenital deafness. This mistake is due partly to the fact that parents are slow to admit the presence of a family defect, and partly to the fact that diseases occurring in very early life are apt to be credited with whatever defect is discovered later. Thus falls, frights, etc., are often credited with having caused deafness which has existed from birth.

In every institution examples may be found of deaf-mute children who have one or two deaf parents or grandparents, and of two or more deaf-mute children belonging to one family. Statistics from British institutions have been fully dealt with in the author's chapter on congenital deafness, "Deafmutism, a Clinical and Pathological Study," but it is interesting to examine statistics so entirely divorced from European influence as those of Japan must be. In the *Report of the Tokio Institution* for 1896, Mr. Shimpachi Konishi, director of the school, says, "Out of sixty-six dumb pupils in this school, there is one pupil whose grandfather was deaf, one whose grandfather's brother was deaf, and one whose father's grandfather was deaf. The number of pupils who inherited dumbness directly from their parents is only three, whilst fourteen dumb pupils were born of parents who married their cousins." Now these statistics are quite parallel with similar ones from British and American institutions. Then take the following instances from the Glasgow records:—

1. In the F. family there are 10 children—5 deaf born, and 5 hearing. The parents hear.
2. In the Ayrshire family there have occurred during the present century 42 cases of congenital deafness; deaf-mute children are still being born into this family, and one is being educated in the Glasgow Institution now. There spring from these statistics, for the fuller consideration of which the author's work may be consulted, these two conclusions:—

1. The congenitally deaf and those related to them should not marry.

2. Intermarriage of blood relations should be strongly discouraged.

The first position—that the congenitally deaf should not marry—is generally conceded by those who work amongst the deaf, but the present arrangements for the education of the deaf, and their management in missions and institutes for the deaf during the period of adolescence, is eminently fitted to encourage union between the congenitally deaf. If not during the school period, at least during the period of adolescence, everything should be done to discourage the association of the deaf and dumb with each other, and the danger of their meeting with those similarly afflicted should be constantly kept before the congenitally deaf by those in charge of them. There are a few bad families, to the members of which it would be well to prohibit marriage altogether. Here is an example. It is taken from the Sheffield *Evening Telegraph* for 26th June 1896:—"At an inquest yesterday on William Earnshaw, fifty-nine, a St. Pancras saddler, it was stated that the relatives could not identify the body, as the wife and sister were blind, deaf, and dumb, and that the four children were deaf and dumb. The deceased was deaf and dumb, and was so when he was married." Now such families as this are responsible for a large number of the deaf mutes in our institutions. I do not think it would be wrong to prohibit marriage to the members of such families. I do not say that all the congenitally deaf should be forbidden marriage, but where the defect is so pronounced that the result is a foregone conclusion, I think the interests of the State should go before those of the individual.

The second position—that blood relations should not intermarry—can hardly be dealt with by legislation, and if the facts were known there should be no need for legislation. On the other hand, prohibition involves no injury to any one. The facts are these. The intermarriage of cousins will emphasise in the offspring whatever defects are characteristic of the family; there is no chance of these being neutralised or dying out as the result of the marriage, as is usually the case when persons previously unrelated get married. Given perfectly healthy cousins with no tendency to any disease, intermarriage would not do harm to the offspring, and might conceivably do good. But as such cousins do not exist, marriage between cousins is often disastrous and is always imprudent.

THE MORBID ANATOMY OF DEAFMUTISM.—It is curious that malformation of the external ear is seldom found in deafmutism which so often is congenital. Probably not even in one per cent of deaf-mute children does such a condition exist. In the middle ear pathological conditions are much more common, but these consist of the usual changes—perforations of the membrane, loss of the ossicles, necrosis of the petrous portion, etc. But, of course, it is not to these that the deafness is due. The acquired deafmutism is almost always due to disease of the internal ear which has spread from the middle ear, and caused inflammatory changes involving destruction of the membranous labyrinth and of the nerve structures which it supports. In a number of cases the danger appears to approach from the side of the brain—meningitis—and more rarely still, the damage to the inner ear is due to a primary labyrinthitis.

In congenital deafness the changes on which the deafness depends are not essentially different from those described above, except in cases which depend on arrest of development or malformation. They consist of obliteration of the normal nervous structures by inflammatory new formations, chiefly osseous. It is not possible to estimate the proportion of cases due to malformation, but it is undoubtedly smaller than was formerly supposed. After a series of years it cannot in many cases be decided whether a structure has been obliterated or has not been developed.

The Testing of Deaf Mutes.—It will be found impossible to apply reliable and accurate tests to very young deafmutes. The presence or absence of hearing may be determined, but the amount of it cannot be ascertained till the deaf child has been so far educated as to understand the nature of the tests. Hence it is not worth while testing deaf children till they have been a year at school. In the interval, of course, the teacher has discovered any cases in which much hearing remains. The best tests are a dinner-bell so arranged that one note may be struck at a time, and the human voice used in the production of vowels and consonants. Bezold[1] of Munich used the whole gamut of the musical scale, and made a very interesting discovery, viz. that in some cases islands of hearing exist in the surrounding ocean of silence or deafness. These islands, of course, represent the less injured pieces of the cochlea, and sometimes include only a note or two, sometimes a half or a whole octave. These observations of Bezold give a very strong support to the Helmholtzian theory of the function of the cochlea, but as the islands seldom coincide with the tones of the human voice, and as the contained tones are seldom very distinctly heard, they will probably be of little use in the practical training of the deaf.

The Distribution of Deafmutism.—The proportion of the deaf to the hearing varies in different countries, but it is governed by causes which operate alike in all countries. The following table gives a review of the deafmutism in various countries. It gives the number of deaf mutes per million living as well as the ordinary rate:—

	Rate per million.		
Switzerland	2452	or 1 in	408
Austria	1307	„	765
Hungary	1263	„	792
Sweden	1023	„	977
Prussia	1019	„	981
Finland	1018	„	981
Canada	997	„	1003
Norway	950	„	1052
Germany (without Prussia)	931	„	1074
Portugal	750	„	1333
Ireland	715	„	1398
India	685	„	1459
United States	659	„	1514
Denmark	650	„	1538
Greece	646	„	1548
France	626	„	1600
Italy	537	„	1862
Scotland	530	„	1885
Cape Colony	525	„	1904
England	489	„	2043
Spain	459	„	2178
Belgium	445	„	2247
Australasia	371	„	2692
Holland	335	„	2985
Ceylon	231	„	4328

The causes which determine these variations in the deaf-mute rate are various. Speaking generally, in prosperous flat countries like England, where the population is well fed and housed, where infectious diseases are carefully treated, and where communication is rapid, the deaf-mute rate is moderate. On the other hand, in a mountainous country like Switzerland, sparseness and stasis of the population make for increase of consanguineous union and an increase of congenital deafness, while the greater difficulty of combating the ravages of the exanthemata makes for an increase of acquired deafness, and the deaf-mute rate tends to be high. Similarly new colonies like Australia have a lower deaf-mute rate than the old colonies like Canada, consanguineous union being less common.

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[1] *Das Hörvermögen der Taubstummen*, 1896.

DIAGNOSIS, PROGNOSIS, AND TREATMENT OF DEAFMUTISM.—When a dumb child is brought to the surgeon, the first thing he should do is to test his hearing. The child is say three years old, and his mother is dissatisfied with his speech. She does not think him dumb. He says Ma-ma and Pa-pa, and the mother thinks this proof that he hears. Further, she has noticed that loud sounds, such as the slamming of a door or the fall of a shovel, startle him. Now the facts are that the child says Ma-ma by imitation, he lip-reads, and he turns at the loud noise, either because he hears nothing but very loud sounds, or because he feels the vibration carried along the floor or wall when the door is slammed. It is generally easy to settle the question of the presence or absence of hearing.

Engage the attention of the child with your watch or knife, and let an assistant slip unnoticed behind the child's back with a gong or a bell, and while it is being sounded watch the child's face. If he hear he will turn, or wince, or wink, or in some way show by his expression that he has been affected by the sound. If he show nothing of this kind he will be too deaf for teaching in the schools for hearing children. The more accurate testing of deaf mutes is interesting and has been referred to, but this test is enough for the child as he is first brought before the surgeon. The *prognosis* in such cases is almost uniformly bad as far as the hearing is concerned. It is best to tell the mother that her child will be a deaf mute, and will require to be educated by special methods. In the meantime she may be made to look upon the development of hearing as a possibility, and she should be encouraged to ply the ears of her child with all sorts of sounds, especially with sounds of all pitches, *e.g.* the notes of the violin, piano, concertina, etc., with the view of encouraging such development. But this is rare. I have seen such growth of hearing in one instance, or rather in two children in one family, but it is not common enough to set before the parents of deaf children as a probability. Apart from the deafness, the prognosis in older children should be based on the evidence of natural ability shown by the child. Congenitally stone-deaf children often make bright pupils. Semi-deaf children who have had their hearing damaged by meningitis and scarlet fever are often dull and make little progress.

The *treatment of deafmutism* divides itself into surgical and educational. Five to ten per cent of deaf-mute children have chronic suppurative disease of the middle ear with or without necrosis of the ossicles or internal tympanic wall when they are sent to school. These cases require treatment on ordinary surgical lines. But a far larger number of deaf-mute children have enlarged tonsils and post-nasal adenoids. Now the removal of these growths will not make the children hear, but if any serious attempt is to be made to make the children speak well, these growths should be removed wherever they are well marked. As deafmutism depends in nearly every case on destruction of, or absence of the internal ear, efforts having for their object the restoration of hearing will necessarily fail. But the question arises whether an attempt should be made to use the partial hearing which so many deaf children have. Now this, like almost all questions connected with the education of the deaf, must be decided with reference to the circumstances and position of the child. If the parents can afford it, the child should have a teacher all to himself, and with the help of conversation tubes, phonograph, etc., everything should be done to teach the child through his auditory nerve. But in a schoolroom, with classes of six to twelve in number, this is clearly impossible. Further, teaching by conversation tube is resented by teachers; they don't like the shouting involved. A few teachers in this country and in America have tried this *accoustic method*, and find that the speech of oral pupils is improved in about 15 or 20 per cent of the cases by this method of teaching. If carefully and persistently used this method may be made a great help to oral pupils. Seldom will it be possible to teach the deaf-mute child by the auditory nerve alone.

On the general question of educational methods it is not the intention of this paper to dilate at length, but it may be stated that in the opinion of the writer the *oral method* should be adopted for the preservation and perfecting of the speech which the semi-mute have still left. Also when there is any great remnant of hearing the oral should be combined with the accoustic method, and in these two classes of cases the semi-mute and the semi-deaf signs should be excluded as much as possible. But there remains the great class of the true deaf mutes—those who have never spoken and who have no hearing which can be used. These form two-thirds or three-quarters of the entire number of the deaf and dumb. When one of these children can be taught in private and have the entire attention of a teacher, the oral method will generally give results gratifying to its parents and of great benefit to the child, and it should only be given up for the finger method if fair progress is not being made with the education of the child. In an institution many children, perhaps most, will do better on the finger method or on a combined method. For the first year the writer thinks all deaf-mute children should be educated on the oral method. At the end of this period the teacher will know if the child is likely to make a good oral pupil, and will regulate his future accordingly. This involves two schools—an oral and a finger school—in every large centre of population, from the former of which all signs are as far as possible excluded. Like the hearing child the deaf mute should be sent early to school, and in this country both law and expediency have fixed seven years as the best time for beginning education at school. For Scotland in 1891 and for England in 1894 an Act came into force, the chief provisions of which are these—Education is compulsory from the age of seven to that of sixteen, and the duty of seeing that this is carried out is laid on School Boards or the school authorities of the parish where the parents of the deaf children reside. The authorities, in carrying out the Act, are at liberty to provide a school of their own, or they may send the child to a certified school or institution outside their bounds, the parents being allowed a reasonable amount of liberty in the choice of a school. Where the parents from poverty are unable to pay the fees, the school authority is required to pay, not only for the education, but also for the maintenance of the child.

A list of trades, etc., in which the deaf and dumb are employed, from the *Report of the Glasgow Mission to the Deaf and Dumb*, 1894-95.

Artist	1
Art Metal Workers	2
Bakers	4
Beltmaker	1
Blacksmiths	2
Carry forward	10
Brought forward	10
Boilermakers	6
Bookbinders	20
Bookfolders	4
Boxmakers	5
Carry forward	45

Brought forward	45
Brass Engravers	7
Brass Finisher	1
Bricklayers	2
Brickmakers	2
Brushmakers	4
Butcher	1
Cabinetmakers	2
Calenderer	1
Capmaker	1
Carpet Designers	5
Caulkers	3
Chairmaker	1
Clerk	1
Compositors	5
Confectionery Worker	1
Cooper	1
Diesmakers	2
Domestic Servants	3
Draughtsmen	6
Dressmakers	10
Dyer	1
Engine Fitters	6
Fancy Boxmakers	6
Fishing Tackle Makers	2
Gardeners	2
Glass Decorators	3
Glass Stainers	2
House Joiners	3
Iron Workers	3
Carry forward	132
Brought forward	132
Jewel Case Makers	2
Jeweller	1
Labourers	15
Lamplighters	2
Lithographic Artists	15
Marble Cutters	2
Mill Workers	4
Moulders	3
Needlewomen	8
Painters	2
Patternmakers	4
Pursemakers	2
Rivetter	1
Saddler	1
Sculptor	1
Ship Joiners	5
Shoemakers	6
Silver Engraver	1
Tailors	16
Ticket Writers	2
Tinsmiths	6
Umbrella Makers	5
Upholsterers	3
Washerwomen	3
Weavers	5
Wood Carvers	4
Wood Engraver	1
Wood Turner	1
Total	253

DUMBNESS FROM IDIOCY.—Dumbness is a necessary result of great deafness happening early in life, and of congenital deafness, but dumbness is no necessary concomitant or result of idiocy. Most imbecile children can be taught to speak. Dr. Ireland says "The lower classes of idiots never learn to speak at all. Out of 103 cases of which I have notes 36 were found mute on entry, and 67 could speak more or less. The average time at which they began to speak was 4 years and 3 months. Only four were noted as having spoken at one year." Children of this class, when they are dumb, are so because they have no ideas to express, and speech develops as education proceeds. At Larbert, Stirlingshire, Mr. Skene, the superintendent of the institution there, showed the writer a cretinous idiot who had not only improved in growth and in general condition very remarkably, but whose speech showed an equally satisfactory development under the use of thyroid gland and extract, and this treatment promises no less brilliant results when applied to cretinous children than it has given in myxœdema. Two classes of idiotic children remain to be mentioned—the deaf-mute idiot and the aphasic idiot. Both of these should be educated as idiots, and not in schools for the deaf and dumb. The aphasic idiot or the idiotic aphasic hears perfectly, but is not intelligent, and does not do well in the schools for the deaf and dumb. The deaf-mute idiot is hardly capable of education in the ordinary sense at all. Most idiotic children have good musical ears, and perform and sing in action songs very well.

LITERATURE.—1. LOVE and ADDISON. *Deafmutism: a Clinical and Pathological Study*, 1896.—2. IRELAND. *The Mental Affections of Children*, 1898.—3. A. GRAHAM BELL. *On the Formation of a Deaf Variety of the Human Race*, 1883.—4. *Bonet Method of Teaching Deaf Mutes to Speak*, translated by H. N. DIXON, 1888.—5. MYGIUD. *Deafmutism*, 1894.—6. FAY. *Marriages of the Deaf in America*, 1898.—7. ARNOLD. *Education of Deaf Mutes*, 1888.

Death, Signs of.—The distinction between the quick and the dead is not always so absolute as might at first appear. Long after what is called the moment of death vital phenomena continue in some of the tissues of the body, and even if we set aside all cases of Indian fakirs and Colonel Townsends, there remains a sufficient residuum of cases of persons presumably dead afterwards returning to life to raise in the minds of some a morbid fear of being buried alive. The conditions which may thus most nearly resemble death are syncope, asphyxia, and trance.

The principal signs relied on as proving that death is real and not apparent are:—

1. *Entire Cessation of Circulation and Respiration.*—The entire cessation of all respiratory movement for a period of five minutes is itself sufficient proof that death is real and not apparent, but the test of auscultation should be applied for a sufficient period to make sure that the action of the heart (*primum vivens et ultimum moriens*) has really finally ceased. The popular method of testing for the continuance of respiratory movements consists in holding a cold mirror or a fluff of cotton before the nose and mouth. If respiratory movements are still going on the mirror will be dimmed, or the fluff of cotton by its movement will betray the passage of the air. To test the cessation of the circulation it has often been suggested that a small vein should be opened. A neater test is that proposed by Magnan, who recommended that a ligature should be tied firmly round a limb. If circulation was going on even slowly the limb beyond the ligature would gradually become congested.

2. *The Cooling of the Body.*—Under ordinary circumstances a dead body is quite cold in from twelve to twenty hours after death. The rate of cooling is, however, modified by many circumstances. Thus a fat body will cool less quickly than a thin one. A body naked or exposed to cold air will cool more quickly than a body wrapped in woollen coverings or surrounded by a warm atmosphere. In water a body will cool more rapidly than in air of the same temperature. It is said that when death occurs from hyperpyrexia the temperature may even continue to rise for a short time after death.

3. *Cadaveric Rigidity.—Rigor Mortis.*—After death the muscular system typically passes through three stages. First, there is a stage of flaccidity in which the muscles retain their power of contracting on electric stimulation. Muscles which were contracted in the act of dying may pass into a condition of cadaveric spasm instead of relaxing at "the moment of death." The second stage is that of rigor mortis. The third is that of relaxation and incipient putrefaction. Rigor mortis usually sets in six or eight hours after death, and may last for three to six or seven days. Its usual duration is about twenty-four hours. It commences in the muscles of the neck and lower jaw, and passes on into the muscles of the upper limbs, chest, and lower limbs. It passes off in the same order. Rigor mortis is a phenomenon analogous to the coagulation of the blood. It is due to the coagulation from the muscle plasma of a proteid termed myosin, and the process is accompanied by the liberation of carbonic acid, and a change from alkaline to acid in the reaction of the muscle substance. The period of onset and the duration of rigor mortis depend chiefly on the degree of exhaustion of the muscular tissue. When death has occurred from convulsions or by accident, for example, during prolonged and excessive muscular exertion, rigor comes on very rapidly and passes off correspondingly quickly.

4. *Hypostasis.*—The gravitation of the blood to the most dependent

parts of the body results in the post-mortem staining which begins to appear in from eight to twelve hours after death. If the body has been lying on a hard table the parts in actual contact with the table, such as the buttocks and scapular regions, may be quite white, while the parts immediately surrounding are deeply stained. Hypostasis must not be confounded with ecchymosis, in which condition the blood is actually extravasated into the subcutaneous tissue. The distinction in a doubtful case is easily made by making a small incision into the discoloured part.

5. The *lustre of the eye* is very quickly lost after death, and the tension of the globe falls so that the cornea feels flaccid. These changes occasionally take place even before death; for example, when death is due to malignant cholera.

6. *The Skin.*—After death the skin loses its normal elasticity. It undergoes striking alterations in colour. The first change is a general pallor of the whole surface. A little colour may, however, be left in dilated venules. Ecchymoses, of course, retain their colour. Later, the discoloration due to hypostasis appears, and this is followed by the colour changes associated with putrefaction.

7. *Putrefaction.*—As rigor mortis passes off the putrefactive changes which end in the complete dissolution of the body make their appearance. The first signs of putrefaction are that the limbs become supple, a faint odour becomes noticeable, and a greenish discoloration appears on the abdomen, and later on the chest, face, arms, and legs. The progress of putrefaction may be limited to a greater or less degree under suitable circumstances by the occurrence of mummification or the formation of adipocere. It is said that putrefaction may be greatly delayed in the bodies of persons who have been arsenic eaters.

LITERATURE.—For references to cases of apparent death see TAYLOR's *Prin. and Practice of Med. Ju.* vol. i.; also JAMES BRAID's *Observations on Trance; or Human Hybernation*, 1850.

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Deformities due to SPINAL CURVATURE *and also* TORTICOLLIS *will be considered in their respective headings.*

Arthrodesis

Definition.—Arthrodesis is an operation designed to fix a joint in a case of entire paralysis of the muscles in the neighbourhood of that joint. Its object is ankylosis either fibrous or osseous, and it is obtained by complete removal of the articular cartilages.

Indications for Operation.—1. When the paralytic condition of the limb is very severe. 2. In poor patients who are unable to provide themselves with apparatus. 3. When an apparatus is hardly tolerated and causes pressure-sores. 4. Not in spastic cases nor in paralytic cases after acute febrile disorders, as paralysis especially in zymotic diseases shows a strong tendency to recovery. 5. When two joints of a limb are hopelessly flail-like it is advisable to fix one or both of them.

Methods.—When the operation is done at the knee or at the ankle, the two joints most favourable, the essential part of the proceeding is the total removal of the articular cartilage in such a way that the bone surfaces are left in contact.

At the Knee.—The usual method is as follows, and is carried out with all aseptic precautions. An incision is made transversely across the joint from one condyle of the femur to the other, and passing across the middle of the patella. The patella is sawn through, and is then turned upwards and downwards. The articular cartilage is removed from the back of the patella. The joint is now fully exposed and treated as follows:—All the articular cartilage is carefully cut away with a gouge, scissors, and sharp spoon or knife. From the anterior surfaces of the tibia and of the femur it is also advisable to take a slice of bone off vertically, so as to fix the posterior surface of the patella denuded of its cartilage to them. The bones are now wired together in such a way that the wire passes through the lower fragment of the patella, then through the tibia, the femur, and through the upper fragment of the patella. The limb is now placed in the straight position, and should be kept immobile by plaster of Paris for at least two months, and after that a posterior splint is sufficient.

At the Ankle.—Considerable difference of opinion exists as to the most convenient incision for the ankle whether it should be anterior, posterior, or external. On the whole, the anterior method gives the most room. Against the anterior incision there are urged the section of the artery with deprivation of the foot of a part of its blood supply, and the difficulty of getting the tendons to unite satisfactorily. But in practice these objections are not serious. The incision is made across the front of the joint from one malleolus to the other, dividing all the soft structures. The joint is thus freely opened, and the articular cartilages completely removed. It is not necessary to dissect away every piece of the synovial membrane, since in those cases in which it has been left its presence has not interfered with the formation of bone. But no loose pieces of cartilage are to be left in the wound, and none attached to the surface of the bone. It is often advisable to freely "stipple" the bony surfaces. The tendons are then united and the wound closed. The foot is placed in plaster of Paris at a right angle for six weeks. By this time firm fibrous union has occurred. To ensure osseous union, a difficult proceeding, the bones have been united by catgut, kangaroo-tendon, or ivory pegs. But absolute osseous ankylosis is not necessary, and is rather to be deprecated at the ankle. Close fibrous union allowing about 10 degrees of movement gives the best possible result. In cases of paralytic talipes calcaneus the best incision is a curved one beginning behind the peronei tendons, running down to the point of the heel and then upwards to the posterior edge of the flexor longus pollicis. The flap so made is lifted up with the tendo Achillis in it, and the opportunity is thus given of shortening the tendon. The joint is then freely opened and the cartilage removed.

Results.—The result of the operation is to render a flail-like limb comparatively secure, and to lessen the number and weight of apparatus required. No fear need be entertained of difficulty in obtaining primary union in these paralytic limbs.

CONGENITAL DISLOCATIONS

Congenital Dislocation of the Hip.—*Etiology.*—It is more common in the female than in the male sex, and is also hereditary. The number of unilateral and bilateral cases is about equal. One important factor in its production is a breech presentation, and if this be combined with a faulty acetabulum then the head of the femur is easily displaced at birth.

Pathology.—The Acetabulum.—In some cases it is entirely wanting, while its site is occupied by a convex mass of bone. In other cases, the cotyloid cavity is present, but is defective. It may be found small and shallow, and about one inch in diameter. In shape it is sometimes circular, but more often triangular, owing to non-development of the ischial part of the Y-shaped cartilage. Its shallowness is also due to defective development of the cotyloid rim, especially at its postero-superior border. The upper extremity of the femur is normal at birth, but as age advances the head undergoes flattening in front and above, and the neck becomes considerably shortened and less obtuse, and further is anteverted or retroverted. The *round ligament* is occasionally thick and solid, but more often elongated and attenuated or even merged into the joint-capsule at the anterior part. In 83 per cent of the cases it is absent. The *capsular ligament* is always elongated, and sometimes is remarkably thickened, while at other times it is seen to be dilated and thinned. Sometimes it is of a curious hour-glass shape, and is obliterated in its middle. As to the *muscles*, the ilio-psoas undergoes such a change in its direction that it finally comes to lie internal and even posteriorly to the head of the bone. The displacement of the caput femoris is usually above and posterior, but as the subject is more carefully studied the number of anterior dislocations is found to be large. The *symptoms* are as follows:—A typical rolling gait, lordosis, prominence of the abdomen, shortness of the lower limbs in proportion to the general bodily development, and feeble development of the muscles of the lower extremity. When the patient stands the great trochanters are unusually prominent, and displaced forwards and upwards or forwards and backwards. The heels also are rotated inwards and the toes turned outwards. When the patient is placed recumbent, the lordosis disappears and the tops of the trochanters are not so near the crests of the ilia as when he is standing. The upper borders of the trochanters are in all cases above Nélaton's line. The limb can often be made to "telescope" up and down. Signs which are absent are: pain and limitation of movement, especially in young children.

Prognosis.—As to the possibility of cure, the earlier treatment is commenced the more likely it is to be successful, and if it be begun before the child is four years of age it probably will be so entirely, but after that the prospect is doubtful no matter what form is adopted.

Diagnosis.—It is essential to make sure that the deformity is truly congenital, *i.e.* has arisen from causes *in utero* and not to unskilful and violent delivery. Paralytic dislocation should also be excluded. Coxa vara is diagnosed from congenital dislocation by the facts that in the former the limb cannot be pulled down by traction, and there is no flexion of the thigh and no muscular wasting. Pseudo-hypertrophic paralysis has often been confounded on superficial observation with congenital dislocation on account of the lordosis and waddling, but a little care suffices to distinguish between them. Tuberculous coxitis has been confused with it, but the pain, limitation of movement, with loss of complete flexion, are quite sufficient for differential purposes.

Treatment.—The difficulties which have to be overcome are: the ill-developed acetabulum, the alteration in the shape of the head, of the femur, and in the direction of the neck, the stretched condition of the capsule, and the contraction of the adductor muscles.

Prolonged rest with extension has been advocated by Buckminster Brown and William Adams, who have published several cases, but it must be confessed that grave doubts have arisen as to the permanency of the alleged cures. The time involved in treatment is at least four years, two of complete recumbency and two of walking on crutches. The disadvantages of this method are evident. Schede has modified this by placing the patient in the recumbent position, applying extension to the abducted limb and moderate lateral pressure on the trochanter. He does this for three months, and then allows the patient to get about on the sound leg with an outside steel support for the affected limb. In this support there is an abduction screw, by turning which pressure may be made on the trochanter. This treatment is suitable only for children under three years of age. Lannelongue, in addition to the rest and recumbency method, has injected two or three minims of a ten per cent solution of chloride of zinc with the object of causing firm fibrous growth around the head of the femur.

Treatment by *reduction*, as initiated by Paci and modified by Lorenz. —The method is as follows. The patient is anæsthetised fully, and placed on a low couch so that the surgeon can stand well over him. The limb is then extended either manually or by means of pulleys until the top of the trochanter is at or just below the Nélaton line. If the muscles refuse to allow the trochanter to be drawn down they are divided subcutaneously. The limb is now fully abducted, and by a little judicious rotation the head of the femur may be felt to slip over the posterior upper margin of the acetabulum, and it sometimes moves in with a distinct click. The difficulty now is to keep it fixed. This is best effected by placing the thigh at about 75 degrees of abduction and using plaster of Paris. In about three weeks the patient is allowed to walk with the leg in this position. Two or three inches of cork-sole are added to the shoe on the sound side, and the assistance of a crutch or crutches is useful. The object of keeping the limb in this abducted position is to wedge it firmly into or upon the acetabulum. The plaster of Paris is renewed at the end of three months and reapplied for a second period.

Treatment by *open operation*. The original method of Hoffa has now been discarded, and Lorenz has modified it. But it is to be noted that when surgeons of great operative skill and experience have endeavoured to follow in Lorenz' footsteps, they have met with nothing like the success he claims. Arbuthnot Lane states that he has had good results by fixing the head close behind the anterior inferior spine.

Other Joints.—Congenital dislocations of almost every other joint of the body have been met with, but they are merely surgical curiosities of no practical interest.

CEREBRAL AND SPINAL PARALYSIS, DEFORMITIES ARISING FROM

The forms of cerebral and spinal paralysis which give rise to deformities are: acute anterior polio-myelitis, of the cerebral and spinal forms; infantile hemiplegia and spastic paralysis of childhood; locomotor ataxy and Friedreich's disease; syringo-myelia; compression paraplegia arising from caries of the spine.

Deformities arising from Acute Anterior Polio-myelitis of Cerebral and Spinal Origin.—The deformities met with in the *trunk* are scoliosis, kyphosis, and lordosis of paralytic origin. Of these the most interesting is scoliosis and this is of an extremely intractable form, but it must be treated by massage, electricity, and supporting apparatus. The deformities of the *arm* arising from polio-myelitis are several. The deltoid is usually wasted, and there is often a paralytic subluxation of the shoulder-joint. In the "fore-arm" type of paralysis of Remak the extensor muscles are paralysed, while the supinator longus escapes. Wrist-drop then results. Occasionally the adductor muscles of the thumb are affected.

When the *lower extremities* are completely paralysed they are small, cold, and bluish, perfectly limp, and swing like a flail in all directions. The joints are so lax that the segments of the limbs may be made to assume almost any position. It is important to ascertain in these severe cases of paralysis of the lower limbs if the psoas have or have not escaped, for so long as the psoas retains power the patient can be made to walk with instruments after tenotomy and when any contraction has been rectified. Paralytic subluxations are either met with in the hip, knee, or ankle.

Treatment of Paralytic Deformities.—In the first place every effort should be made from the time of onset of the paralysis to retain the limbs in as nearly normal a position as possible. That is to say, it is important to anticipate the deformities. Electrical stimulation of the muscles, friction, and massage should be assiduously persevered with. The active treatment of these deformities must be of two kinds; mechanical and operative. The objects of mechanical treatment should be to support and protect the paralysed limb in such a way that the muscles shall work to the best advantage, and that the joints are supported and controlled; also to overcome by means of suitable apparatus deformities which have already occurred, and to prevent their recurrence. The operative treatment consists in tenotomy and fasciotomy, osteotomy, arthrodesis, and tendon-transplantation. Tenotomy is useful in relieving the various forms of talipes of the foot, the contraction of the knees and the flexion of the thighs. Osteotomy is not often called for. The indications for arthrodesis have been given in the article of that heading, and the indications and methods for tendon-transplantation are treated under that title (p. 432).

Infantile Hemiplegia and Spastic Paralysis.—Deformities of a most persistent and intractable nature often ensue from these causes, and the arm is more affected than the leg. The position assumed by the arm is quite typical. In this limb flexion predominates. The shoulder is sometimes raised and sometimes lowered. The upper arm is generally kept parallel with the trunk. The forearm is flexed at a right angle, and is generally in apposition with the lower part of the chest or the upper part of the abdomen. It is almost invariably pronated, very rarely supinated. The wrist is strongly flexed, and the hand is adducted. The fingers are firmly contracted. The leg is in a condition of extension, and the foot assumes a talipedic form, either of equinus or equino-varus. The mode of walking is thus: the heel is raised and the foot is lifted from the ground with difficulty, the toes scrape along the floor and later the patient becomes scissor-legged in progression. The treatment is as follows:—Every effort must be made, if the case be seen early, to prevent the onset of deformity by massage, manipulation, passive movements of the joints. The patient should also be taught to use the affected limbs as much as possible, and much improvement may be expected in slighter cases. With reference to the use of retention apparatus, the writer's experience is that directly it is removed the spasm returns. With regard to operative measures in the upper extremity, the writer has lately devised and successfully carried out an entirely new form of operation. It is as follows:—The tendon of the pronator radii teres is exposed and separated from its insertion into the radius. A gap is made in the interosseous membrane, and the pronator radii teres is carried round *posteriorly* to the radius, and is reinserted at its normal site. It therefore becomes a supinator instead of a pronator. The flexor carpi radialis and other tendons at the wrist are divided so that the wrist drop is overcome, also the contraction of the fingers. In the lower extremities, operative measures are fully indicated. For spastic talipes the tendo Achillis should be severed, for contracted knees the hamstrings, and in the thighs, the tensor vaginæ femoris, the sartorius and adductors should be divided. It is astonishing the amount of good that results from this proceeding.

Locomotor Ataxy.—This causes spinal deformities very rarely. Sometimes the deformity is angular, or sometimes scoliosis is present. In Friedreich's disease or congenital locomotor ataxy, scoliosis develops late in the disease, and is generally seen to be with the curve to the right in the dorsal region. In this disease, talipes arcuatus is also met with, and is spoken of later. Progressive muscular atrophy gives rise to a peculiar form of talipes varus, due to peroneal paralysis. In syringo-myelia various distortions of the spine may be met with, and many forms of contractions of the limbs.

Compression paraplegia is a frequent complication of spinal caries. It is rarely due to pressure on the spinal cord by bone, but more frequently it is caused by a tuberculous pachymeningitis, the thickened membranes constricting the cord. The paralysis is usually bilateral. In rare cases it is unilateral. It affects the legs generally, although the arms may suffer later, or both may be paralysed simultaneously. It occurs in about 1 in 12 to 15 spinal cases. The symptoms of compression paraplegia are as follows:—Its onset is in some cases sudden, but more often it is gradual. As to the motor functions, the patient complains of getting tired easily, and soon the legs begin to drag and the toes to catch in walking. Dull aching pain is common in the early stages in the body and limbs. In the body the most usual form is girdle pain or pain in the pit of the stomach. These pains are due to irritation of the nerve roots. Oftentimes there is no anæsthesia, or it occurs occasionally without motor symptoms. The reflexes are exaggerated both superficially and deeply. The sphincters are often involved, and incontinence of urine and fæces occur in severe cases. The affected muscles waste, and the reaction of degeneration is more or less marked. The limbs are often cold, but sometimes perspire persistently. The diagnosis is not as a rule difficult when the back is examined. Sometimes it happens that compression paraplegia sets in before deformity has appeared. The prognosis of these cases is that a great number of them show a strong tendency to complete recovery without operation. Mere recumbency with extension is often sufficient to induce an immediate change for the better. Recurrent attacks are dangerous but not so *per se.* Cystitis and bronchitis are grave complications. With regard to treatment, prolonged recumbency with extension of the spine in the horizontal position often effects a surprising improvement, and should be persevered with. But if after 18 months to 2 years of this treatment the symptoms do not improve but rather get worse, then laminectomy should be resorted to. Too great results must not be expected from laminectomy, for the operation is as often a failure as it is a success. Forcible rectification of the deformity has relieved some troublesome cases of paralysis.

RICKETS—DEFORMITIES OF

The skull is often enlarged and thin, and with it there is associated hydrocephalus. The fontanelles remain open. The forehead is square and the upper wall of the orbit is oblique. There is also a peculiar development of the lower jaw; its alveolus is somewhat inverted, so that the teeth point inwards. With regard to the spine, the rickety deformities are kyphosis and, later, scoliosis. The chest, too, is the subject of deformity, pigeon-breast, exaggeration of the curves, and subluxation of the inner end of the clavicle. Rickety deformity of the arm is sometimes seen in severe cases, and both bones of the forearm may be bent inward or outward, or one bone alone. The rickety pelvis is important to obstetricians, and the conjugate diameter is decreased owing to the prominence of the sacro-vertebral angle. In consequence of the inward thrust of the head of the femur into the acetabulum on each side, the lateral aspect of the pelvis is flattened, and the pubic arch diminished. The rickety deformities of the long bones of the lower extremity are fully described in the sections dealing with deformities of the knee and foot.

Tendon-Transplantation

By this is meant the reinforcement of a paralysed muscle by attaching to it the tendon of a healthy muscle. It is also known by other names, as muscle-grafting, function transference of tendon, but the word tendon-transplantation sufficiently indicates the scope of the procedure. Before deciding to perform tendon-grafting, there are certain points to be observed, namely:—1. The muscles should be carefully tested electrically, and the relative strength of each of them determined; 2, all conditions due to contraction of the plantar fascia, such as pes cavus, should be previously removed by operation; 3, the operation is rarely called for when one muscle only is partially paralysed; 4, cases in which all the muscles are paralysed are suitable for arthrodesis only; 5, in selecting a healthy muscle for reinforcing a paralysed muscle it is advisable that the one selected should belong to the same group, if possible, as the paralysed one, and the muscle selected should also be as far as may be, in the same line as the paralysed muscle; 6, the reinforcing tendon should be carried as directly as possible to the paralysed muscle and not bent round at an angle. Tendon-transplantation is particularly applicable to talipes calcaneus, as the peronei muscles and the posterior tibial deep muscles, *i.e.* the tibialis posticus and the flexor longus digitorum and the flexor longus pollicis are very suitable for grafting into the tendo Achillis. Cases of simple equinus do not as a rule call for muscle-grafting, because division of the tendo Achillis is sufficient. Cases of paralytic valgus due to paralysis of the tibialis anticus and posticus may be treated by transplantation of the extensor proprius pollicis into the tibialis anticus and a piece of the flexor longus digitorum tendon into the tibialis posticus. On similar lines talipes varus may be treated. As to the actual method of procedure, the operation requires that the parts should be absolutely aseptic. The tendons to be operated upon are exposed, and their sheaths freely opened in such a way that the tendon of the reinforcing muscle can be applied easily to the paralysed muscle. Taking *e.g.* the fixing of the peroneus longus to the tendo Achillis, the sheaths of the peronei and of the tendo Achillis are freely opened up and a considerable portion of the peroneus longus tendon is detached. It is then divided across just above the malleoli and passed to the under surface of the tendo Achillis nearly opposite the ankle joint, the tendo Achillis having been previously split longitudinally at this spot. The proximal end of the peroneus longus tendon is drawn through it and split somewhat, and the ends attached to the posterior surface of the tendo Achillis by being sewn down with silk or some other material. The proximal part of the tendon of the reinforcing muscle should be pulled quite tightly, and inserted at such a spot in the tendon of the paralysed muscle that the former, when it is fixed, is at its utmost possible tension. The wound is then allowed to heal and the strength of the reinforcing muscles is subsequently increased by massage and galvanism.

Hand and Fingers

Congenital Deformities.—These comprise *club hand*, *congenital contraction of the fingers*, *supernumerary fingers* or polydactylism, *suppression of the fingers*, *web-fingers* or syndactylism, *hypertrophy of the fingers*, *congenital lateral deviation of the fingers.*

Club Hand.—This is a very rare deformity, and there are comparatively few cases recorded in literature. In most cases the child is both premature and still-born. The forms of club-hand are as follows, and it should be remembered that the hand may deviate either to the outer or inner border of the fore arm, or be in a position of flexion or extension. Hence we have radial and ulnar club-hand and palmar and dorsal, so that there are the radio-palmar, radio-dorsal, ulnar-palmar, and ulnar-dorsal. In the palmar cases the hand forms with the forearm a more or less acute angle open anteriorly. The lower end of the radius is prominent posteriorly, and the carpus articulates with the anterior surface of the radius. There is generally some degree of mobility of the hand on the forearm. It is small and wasted owing to the shrinkage of muscles and the absence of some parts of the bones.

Treatment.—The means which are adopted to alleviate the deformity are passive movements, massage, the use of retentive apparatus, tenotomy, and operations on the bones. In any case the result is not very satisfactory.

Congenital Contraction of Fingers.—This deformity is quite distinct from contraction of the palmar fascia known as Dupuytren's contraction, and should not be confused with it. The affection is generally limited to the fifth finger, but at times the ring finger and even all the fingers are contracted. It is often associated with congenital hammer-toe, and in that event the second toe is the one usually affected. Some contraction of the finger is frequently met with, but it is only when it gives rise to pain and annoyance that any notice should be taken of it. In congenital contraction the first phalanx is usually hyper-extended, and the second and third are flexed, in contra-distinction to Dupuytren's contraction, in which the first and second are flexed and the third is generally extended. If the deformity is left unrectified, contraction of the skin and fascia and of the lateral ligaments of the inter-phalangeal articulations takes place. But the contracted fascia never extends up into the palm as in Dupuytren's contraction.

Treatment.—In the milder degrees it is sufficient to straighten the affected fingers by frequent passive movements, and to maintain the correction by the use of a small malleable iron splint adapted to the dorsal surface of the fingers. This often suffices to remove the deformity. Should it fail to do so, the contracted fascial bands may be divided and the finger put up in a suitable splint. Unfortunately these cases have a strong tendency to relapse, and it is well to warn the patient that after a finger has been straightened a long course of mechanical treatment is necessary to maintain the improvement. For this purpose the apparatus similar to that used after operation for Dupuytren's contraction must be worn day and night for three months.

Supernumerary Fingers—Polydactylism.—There are five varieties of polydactylism. 1. An additional finger is more or less developed, generally on the ulnar border of the hand, being attached to it by a narrow pedicle.

2. An additional thumb is more or less developed. 3. A supernumerary digit more or less perfect is closely united throughout its whole length to another digit. 4. A completely developed extra digit is formed, and possesses its own separate functions and tendons. 5. The bifurcated hand which has eight fingers and no thumbs.

Treatment.—In the first variety the additional digit should be removed early in life. In the second variety removal is indicated too. As to the third variety the propriety of operation is doubtful. In the fourth variety it is advisable not to interfere, and the fifth admits of no treatment.

Suppression of the Fingers.—These are of interest rather to the teratologist than to the surgeon. If the hand is very unsightly or useless, amputation of it is the best resource, and a good artificial hand is to be preferred.

Webbed Fingers or Syndactylism.—There are three varieties. (1) Two fingers, generally those on the inner side of the hand, are united by skin and fibrous tissue. (2) The union is by muscular as well as fibrous tissue and skin. (3) The bones are fused throughout their whole length, or more often at the second and third phalanges only.

Treatment.—In the second variety little can be done, and the case is often best left alone. In the first and third varieties the chief difficulty after operation is to prevent some re-formation of the web, especially towards the base of the new cleft. This difficulty can be overcome either by the formation of a permanent opening at the bottom of the web by transfixing it with a silver pin or by Didot's operation, or by those operations known by the names of Zeller and Norton. Choice of operation.—1. If the web is small and thin, or if the union between the fingers is very close, the formation of a permanent opening at the base of the web is to be preferred. The web may be divided subsequently, and its edges trimmed and sutured. 2. If the web is extensive, complete, and of good width, Didot's operation is the best. 3. If the web is incomplete and reaches but halfway, Zeller's or Norton's operation is indicated.

Hypertrophy of the Fingers.—This condition is sometimes seen at birth in a minor degree, and becomes exaggerated later. The hypertrophy may consist of general overgrowth of all the tissues of the finger, lymphatic enlargement of the subcutaneous tissue, or a nævoid condition of all the soft structures.

Treatment—Compression of the fingers and ligature of the arteries have been both tried, but without success. When the finger becomes a source of annoyance it should be removed.

Congenital and Lateral Deviation of the Fingers.—This is very rare indeed, but is comparable to a condition seen in the toes. It may be treated either by a suitable apparatus, or by division of the lateral ligaments, or by amputation.

ACQUIRED DEFORMITIES.—Under this heading are comprised *Dupuytren's contraction*, *spring finger*, and *mallet finger.*

Dupuytren's Contraction.—*Definition.*—A permanent flexion of one or more fingers arising from contraction of the palmar fascia and its digital prolongations.

Occurrence and Etiology.—It is many times more frequent in men than in women. As a rule the ring and little fingers are affected, but it may spread to other fingers. In some cases it is distinctly hereditary. The age at which it comes on is generally in middle or in late life, that is, when fibroid changes supervene. Occupations seem to have some share in its production, especially those that involve repeated traumatism to the palm of the hand, as in gardeners, drivers, engravers, etc. In some instances it would appear as if the contraction began after a definite injury to the palm—a slight wound, a sudden bruise, or an excessive strain of the structures. Certain general conditions seem to predispose to it as, for instance, gout and rheumatism, or the contraction appears to be much more common in gouty and rheumatic people, and to follow sometimes an attack of gout. Other people have noticed it to be associated with syphilis, and Mr. William Anderson has hazarded the opinion that it is of bacterial origin.

Its *morbid anatomy* is as follows:—The affection is primarily a contraction of the fascia, and secondly of the skin. The tendons have nothing to do with it. The palmar fascia is not a well-defined aponeurosis, but fades off gradually at its edges and gives off two sets of processes, the superficial to the skin and the deep to the lateral aspect of the fingers, passing to the sides of the first and second phalanges, and to the periosteum and to the tendon sheaths. The nature of the morbid change in the fascia is a fibroid hypertrophy. Sometimes this appears in the form of small fibromata, in others there is a general thickening of the bands, followed by contraction. Crystals of urate of soda have been found in the thickened fascia.

Symptoms.—At first there is a feeling of tightness in the palm of the hand and in the ring or little finger, and the patient finds some difficulty in fully extending the fingers. Later there appears nodular indurations with adhesions of the skin. The latter is first seen usually in the transverse crease of the palm. The affected fingers then begin to retract, and the first phalanx flexes on the metacarpal bone.

Treatment.—No measures except operative are of any avail. The operative measures are either multiple subcutaneous division of the contracted band as practised by Mr. Adams, or open removal by a careful dissection of the palm of the hand. The method to be preferred is the multiple subcutaneous puncture. The operation may have to be repeated two or three times. After the operation a Dupuytren's splint is worn, and the fingers are gradually straightened. The result is as a rule very good, and relapses are not frequent. The writer has performed the open method several times in hospital patients, who cannot afford time to attend frequently, and he has been satisfied with the result.

Spring Finger, known also as *Jerk or Snap Finger*. The description of this affection is that if the patient closes all the fingers on the palm, on opening them he finds that one remains shut, and it can only be extended by the other hand, and flies open like a knife-blade with a snap. Sometimes there is difficulty also in flexing the finger, which is accompanied by a small jerk. The affection is usually seen in the thumb, and is due to one of two causes, either a thickening of the tendon as it passes through its osseo-fibrous groove, or else narrowing of the groove. *Treatment.*—If the thickening can be felt it should be cut down upon, and the thickened portion removed. In many cases, however, it is sufficient to blister frequently and the thickening disappears, or the finger may be fixed in a metal splint with pressure over the spot where the movement of the tendon is hindered.

Mallet Finger.—This is also known as "drop finger," and is due to subcutaneous rupture of the extensor tendon where it is inserted into the last phalanx. Its usual cause is a fall forwards on the fully extended fingers, with the result that the fibres of the extensor tendon are torn away and the finger cannot then be fully extended With regard to treatment, the finger may be placed in a malleable iron splint, with the last phalanx hyperextended; or an incision should be made over the dorsum of the last phalanx, and the tendon reunited to the bone.

Hallux Valgus (Bunion)

It is largely due to the use of improper boots, not necessarily of light ones, but of those which are pointed and often too short. In some cases it is due to osteo-arthritis and gout. The deformity is a displacement of the great toe outward, with prominence of the base of the proximal phalanx and of the head of the first metatarsal bone, mostly of the latter. Both these portions of bone are often enlarged and covered by a bunion or bursa. The bony swelling is especially noticeable in cases of osteo-arthritis. The anatomy of the affection is a partial subluxation of the first phalanx outwards from the head of the first metatarsal bone, leaving it exposed to pressure. The ligaments on the inner side of the joint are stretched, and occasionally perforated; those on the outer side are shortened. Of the tendons the extensor proprius pollicis is displaced outwards. The formation of the false bursa or bunion is secondary to the enlargement of the bone. The bunion is very liable to inflammation, and suppuration may cause cellulitis with occasional disorganisation of the joint. Sometimes the bunion has a corn on it. The symptoms are sufficiently plain, but the affection varies in degree, and there is no doubt that women suffer more frequently than men.

Treatment—Prophylactic. Pointed boots must be absolutely forbidden, and the inner edges of the boot should be quite straight. In slight cases curative treatment consists in wearing proper boots, with the application of cold and soothing lotions to the inflamed and thickened skin, and the wearing of the digitated or divided socks. Some advise the use of a post between the first and second toes in the boot, but this does not answer well in practice. In other cases a bunion spring is used, but if the deformity is at all severe and the bursa large and painful, the only course is operation. It is not sufficient merely to chisel away the prominent portion of bone, but the operation *par excellence* for these cases is excision of the head of the metatarsal bone. With this operation the writer has ample reason to be satisfied.

Hallux Varus or Pigeon-Toe is the reverse of hallux valgus, but is not associated with pain in the same way. If it is excessive the treatment consists in manipulation and the use of a light splint to press the toe outwards.

Hallux Rigidus is a somewhat curious affection. The great toe cannot be extended beyond the straight line, and any attempt to do so produces severe pain. The tendon of the extensor proprius pollicis is always tense. With regard to the pathology of this affection opinions differ, but the writer has been able to ascertain that it is frequently associated with osteo-arthritis and with inflammation of the synovial fringe between the sesamoid bones. The mechanism of the affection is easy to understand. When an attempt is made to extend the toe, the head of the metatarsal bone and the base of the first phalanx press upon this inflamed spot so that the rigidity of the toe is really reflex. Local measures avail but little, and in cases which are at all marked it is always best to excise the head of the first metatarsal bone and to remove a small portion of the prominent part of the base of the first phalanx.

HAMMER-TOE

Definition.—A deformity usually affecting the second toe and consisting of dorsi-flexion of the first phalanx, plantar-flexion of the second, and extension of the third.

Etiology.—In some cases it is congenital and is associated with congenital contraction of the little finger. In other cases it is distinctly hereditary. But it is sometimes due to acquired causes. The chief of these is the use of short boots. The appearances presented are as follows. On the dorsal aspect of the first inter-phalangeal joint a painful corn is frequently present; beneath this is a bursa which from time to time inflames and suppurates. On the under surface the skin is contracted, and at the bottom of the groove the long flexor tendon can be felt. The first phalanx is in a state of extreme dorsi-flexion, so that the head of the metatarsal bone is uncovered below to about half its extent. The anatomy of the part is as follows. In the first place there occurs contraction of the extensor tendon. This is followed by contraction of the flexor tendon, and with gradual shortening of the lateral ligaments. The latter point is most important to recognise, as upon it depends successful treatment.

Treatment.—In slight cases attention to the boots, together with manipulations and the use of the malleable iron splint at night will remedy the trouble. But in many cases it is necessary to operate, and the writer would decry amputation of the toe, as it is quite possible to remedy the trouble without this rather severe procedure. If the toe be amputated, then bunion almost inevitably follows, because the first toe is readily displaced outwards. The operation consists in subcutaneous section of the flexor tendon and of the lateral ligaments from the under surface of the first interphalangeal joint, and often of the extensor tendon on the dorsum. After the operation the toe is fixed to a malleable iron splint until the small wound is healed, and it is then gradually extended. Good broad boots should be ordered for use afterwards.

TOES—DEFORMITIES OF

The other affections which are met with in the toes are *Syndactylism*, *Polydactylism*, *Suppression of the Toes*, and *Lateral Deviation of the Toes.* The latter is the result of bad boots, and may be remedied by manipulation, the wearing of a digitated sock, or by the use of a sole-plate with slots in it. In moderately severe cases the lateral ligaments may be divided, and in extreme cases amputation is necessary. Hypertrophy of the toes, generally of the first toe, is seen from time to time. The hypertrophied toe is frequently displaced inwards or outwards. It may attain an immense size. In most cases partial or complete amputation is required eventually. The treatment of syndactylism in the toes is the same as that of the fingers, although it may be better to amputate at once.

CLUB-FOOT—CONGENITAL

Etiology.—The causation of congenital club-foot is still doubtful, although much light has been thrown upon it by the labours of R. W. Parker. The matter may be discussed under four headings: (*a*) arrested development of the bones of the leg; (*b*) causes arising from nerve lesions; (*c*) mechanical causes due to malposition of the fœtus *in utero*; (*d*) abnormal development of the bones of the foot. (*a*) In describing bow-legs it is stated that certain forms of congenital bow-legs are accompanied by talipes equino-varus. Again, some cases of varus and equino-varus exhibit congenital absence of the tiba. But these congenital abnormalities are found in only a few of the cases of equino-varus, and cannot be the sole cause. (*b*) Nerve lesions. In some cases meningocele, encephalocele, hydrocephalus and spina bifida co-exist with congenital club-foot, but these are very few in proportion, so that the nerve theory fails to explain the majority of the cases. It has been held by some that the deformity is due to partial asphyxia at birth, resulting in convulsions, but in the absence of definite examples carefully collected and impartially examined, this opinion cannot be advocated. (*c*) Mechanical causes due to malposition or compression of fœtus *in utero*. There is ample evidence to show that this is the probable explanation, and from the figures given by Parker in his work it is easy to understand that owing to intra-uterine packing the foot may during fœtal life be retained in one position which becomes fixed. As a rule the position is such as to lead to some small degree of equino-varus, even in presumably normal infants, and they subsequently lose this tendency to eversion when they commence to walk. If the position is more decided, then an abnormal condition of the foot arises—either equino-varus in both feet, or equino-varus in one and equino-valgus in the other, or calcaneus in both. The most common modes of production are by accidental locking of the parts, by locking of the parts due to abnormal positions of the limbs, by exceptional positions of the limbs, independently of locking, and by congenital absence of certain bones. The last supposed cause (*d*), abnormal development of the bones of the foot, cannot clearly be established; one can only say that the normal inversion of the lower limbs *in utero* persists some time after birth. The reason of the persistence is probably close intra-uterine packing, and this throws us back upon Parker's observations.

Forms of Congenital Club-Foot.—Varieties.—The most common form is TALIPES EQUINO-VARUS. Some authors state that talipes varus is more common, but if a careful examination be made of a large number of cases it will be found that most of the so-called varus-cases have some shortening of the tendo Achillis and raising of the heel, so that really they are cases of equino-varus. And this point is proved by treatment, for until the tendo Achillis is divided, the foot cannot be fully rectified. The next most common form is calcaneo-valgus, while the pure calcaneus and pure equinus are rare; the last-named infinitely so. In the consideration of congenital club-foot, therefore, talipes equino-varus will be described fully.

Appearances.—The nature of the deformity is as follows: The heel is raised and the foot is extended, that is, it is in a position of plantar flexion. The sole of the foot and the toes are adducted and brought to the middle line instead of being directed to the front. The internal border of the foot is raised to a varying degree and bent upon itself, so that it is concave. With this the adduction of the great toe is often very marked. The external border of the foot is convex, and is in contact with the ground and forms the main point of support in progression. The deformity is situated in two places: in the foot chiefly in the neighbourhood of the medio-tarsal joint, and to a less degree at the tarso-metatarsal and phalangeal articulations and at the ankle. Due recognition of the sites of deformity is essential to proper treatment; for the back part of the foot, that is, behind the medio-tarsal joint, must serve as a fixed point for the correction of the front part; and fixation of the back part can only be obtained by leaving the tendo Achillis intact until the front part is restored. Congenital club-foot is more frequent in boys than in girls, and occurs about once in one thousand births. Double congenital valgus is more usual than single. Very frequently with club-foot of congenital origin, meningocele, spina-bifida, partial or complete, amputation of the limbs, absence of the fingers, polydactylism, syndactylism and absence of the bones of the leg or foot are found to co-exist.

Degrees.—The deformity varies according to age. FIRST DEGREE—The foot can be momentarily replaced by manual force. SECOND DEGREE—The foot cannot be replaced manually. On attempting forcible reposition there remains some adduction or extension of the foot, and the sole cannot be planted squarely on the ground, and the great toe is much separated from the second. THIRD DEGREE—Seen in children and adults, the foot is in a rigid and resistant state with aggravation of the deformity and strong contraction of the soft parts. FOURTH DEGREE—The deformity is inveterate and of old standing, and much malposition of the bones is present with the thickening of the soft tissues, and formation of false bursæ on the outer side of the foot over the bony prominences. The foot may be so much distorted that the patient walks on the dorsum, and the sole of the foot looks upwards and backwards.

MORBID ANATOMY.—Authors are agreed that in talipes equino-varus there exists extension of the astragalus upon the leg at the ankle joint with twisting downwards and inwards of its head and neck, and inward subluxation of the scaphoid, elevation of the tuberosities of the os calcis, and rotation of the same bone around its vertical axis. The bones are modelled first of all in cartilage, and so long as they remain unossified they may still be moulded to a normal shape and no extensive operation is needed. Without discussing the minutiæ of the morbid anatomy of club-foot it is necessary to remark that the angle of the neck of the astragalus is increased from thirty-eight to about fifty degrees. An astragalus taken from an adult case of club-foot presents several new features. Its body is no longer square but trapezoid, and even triangular, and the head protrudes from the front of the ankle-joint. The scaphoid is found to be much atrophied and its tubercle has very nearly disappeared. On the anterior aspect of the internal malleolus is a new facet for articulation with the scaphoid. The ligaments on the dorsum and external border of the foot are elongated, and those in the sole and on the internal border are contracted. The plantar fascia is also contracted. The tendons at fault are the tendo Achillis, tibialis anticus, and posticus, extensor proprius pollicis, the flexor longus digitorum, and the flexor longus pollicis. Other somewhat remote lesions are met with, associated with congenital club-foot. One interesting lesion is genu recurvatum with absence of the patella, another is scoliosis.

The Obstacles to Reduction in Congenital Talipes Equino Varus arise (1) in the infant, from the altered direction of the neck of the astragalus, the contracted anterior fasciculus of the internal lateral ligament, the contracted astragalo-scaphoid and calcaneo-scaphoid ligaments; (2) in the adult from the profound alteration in the shape of the bones, from the formation of new joints, from fixation of the ligaments and tendons in their abnormal attachments and course.

Prognosis.—The points in any given case on which information will be sought are the following: (*a*) Can a perfect foot be obtained? (*b*) Will a shapely foot result from treatment? (*c*) Will the patient be able to walk comfortably and rapidly? (*d*) What possibility is there of relapse, and if relapse occur, can the foot again be rectified? (*e*) The duration of treatment? The answer to question (*a*) depends upon the age at which treatment is begun and the degree of deformity. Many cases of congenital varus of the first and second degrees are cured if treatment is begun before walking is attempted. Cases of the third degree, especially after weight has been borne on the deformed feet, seldom give perfect results. In any event, persistency in treatment is essential, and it is the duty of the surgeon to insist that relapse is a likely event so long as growth is going on, and every care must be taken to prevent it. (*b*) A shapely foot will often result from early and continued treatment, but in some instances there will always remain a square-toed appearance and the foot is ungainly. (*c*) The question of comfortable walking depends upon the absence of cicatricial material after treatment. Tarsectomies and such like procedures should therefore be avoided if possible. (*d*) As to the possibility of relapse, there is every likelihood of it in congenital cases unless persistent care is exercised all through childhood and adolescence, but relapsed varus is capable of much improvement by the methods to be detailed subsequently. (*e*) The duration of time required for treatment depends on the degree of deformity and the method adopted. The slower orthopædic methods are effectual but tedious. A deformity of moderate severity can be reduced by manipulation and wrenching in two to three months.

Diagnosis.—Some difficulty will arise in distinguishing in a child congenital and paralytic equino-varus, but the main points are, that in paralytic equino-varus the limb is wasted, cold, and blue, and the electrical reactions are entirely lost in the affected muscles. From spastic paralysis the diagnosis of congenital equino-varus is more difficult. The presence of rigidity of the knees, adduction of the thighs, flexion of the forearm and contraction of the hand, are points sufficiently distinctive of spastic paralysis. The hysterical form of equino-varus readily disappears under an anæsthetic.

Treatment.—Of slight cases or cases OF THE FIRST DEGREE: The feet in this degree can be brought to a straight line with the legs by manipulation, and sometimes everted. But when the pressure is relaxed they spring back to their original position. Also they cannot be fully dorsi-flexed when they are straightened and everted. Such cases can be treated by manipulation alone or by manipulation combined with massage and retention apparatus. In manipulation the movements to be practised are abduction and eversion at the transverse tarsal and sub-astragaloid joints, and flexion and extension of the whole foot of the ankle finishing up with circumduction. The movements should be easy and gradual, and should last about five to ten minutes three times a day. Manipulation can be combined with retention apparatus in the following way. A straight well-padded piece of soft iron is bent to the shape of the deformed foot and applied to the outer border of the limb. Gradually the angle of the splint is diminished until the foot can be fixed in a straight line with the leg without pain. This suffices to remove the varus. The equinus can be overcome by the use of a tin shoe with a quadrant movement at the ankle. In place of the splints many surgeons prefer to use plaster of Paris frequently changed, additional rectification being obtained whenever a fresh plaster of Paris splint is put on; this should be at least every third day.

OF THE SECOND DEGREE.—Here the foot can neither be fully everted nor brought into a straight line with the leg. In attempting to do so the tendons of the tibialis anticus and posticus and the flexor longus pollicis, together with the tendo Achillis, become tense. Cases of this degree may be cured by (*a*) tenotomy with the after-use of shoes and apparatus, or (*b*) tenotomy followed by wrenching on two or three occasions and putting the foot in plaster of Paris after each partial correction. As this degree is usually found in children under four years of age, the bones and ligaments are still elastic, and it is not necessary to resort to deep operations such as Phelps' operation or the various kinds of tarsectomy. The following tendons need division. In the first stage, the tibialis anticus and posticus, the plantar fascia and the anterior fasciculus of the internal lateral ligament of the ankle. As to whether reposition of the front part of the foot should be aimed at immediately after tenotomy or gradually is a matter of somewhat diverse opinion. As a rule gradual reposition by means of the flexible iron splint is to be preferred, and the varus will be gradually overcome until the foot is in a straight line with the leg. Then and only then should the tendo Achillis be divided to reduce the equinus. After the tendo

Achillis has been divided an Adam's varus splint, or a tin shoe with a quadrant at the ankle, is substituted for the flexible iron splint. As in the first degree, some surgeons prefer to use plaster of Paris instead of splints or shoes; but its disadvantages are that its use involves a good deal of trouble if it is renewed sufficiently frequently, and the foot cannot be manipulated while it is on. This is a very essential part of the proceedings. After-treatment, extending over some years, by means of walking apparatus, is required. In some of these cases there is very considerable inversion of the whole limb. This may sometimes be overcome by frequent twisting movements on the part of the attendant or by a linear osteotomy.

OF THE THIRD DEGREE.—Rigid or resistant club-foot. Here all the deformities are exaggerated and the bony prominences are well marked on the outer side of the foot and are the sites of considerable pain. The best form of treatment for these rigid feet is, in the writer's opinion, a gradual one. There is no course that answers so well as the following. Take the patient off his feet for two or three weeks and give him complete rest; by so doing the painful spasm of the muscles passes away and the foot soon becomes less rigid. The degree of suppleness which returns to the foot is surprising. Tenotomy of the tibial tendons and plantar fascia is now performed, and the varus is overcome by using a malleable iron splint or by employing a Scarpa's shoe. Happily successful as treatment on these lines is, the length of time occupied has induced surgeons to devise other means of overcoming the difficulties and restoring the foot. A valuable adjunct to treatment is forcible rectification or wrenching. The best apparatus is Thomas' wrench. It is better in obstinate cases to employ it frequently and moderately than with violence. The more extensive operative measures are free subcutaneous division of all the resistant structures at one sitting, Phelps' operation, tarsal osteotomy and tarsectomy. As to the value of either of these operative procedures it is well to remark that Phelps' operation or treatment by open incision has been for some time on its trial and has not given satisfaction. Free subcutaneous section is followed by so much scarring that the foot remains rigid although it may be of a good shape. Tarsal osteotomy is often insufficient because it is difficult so to plan the incisions as to allow of the bones being placed in their proper axes. Of the many forms of tarsectomy it may be said that removal of the astragalus is the best. Now, in commencing the treatment of a case, either in this degree or the next, it is important to decide at once if the foot is capable of reposition by the gradual method, or requires tarsectomy. If the latter measure is called for, the writer has no hesitation in pronouncing astragalectomy to be the operation which is likely to yield excellent results on the following grounds, the resulting good movement of the ankle, the immediate and permanent correction of the deformity, the absence of recurrence and the ease of the operation. As to wedge-shaped tarsectomies, the operation is easy, and it is very tempting, if a foot is too short on the inner side and too long on the outer, to saw out a piece of bone and put the foot straight. But this does not appear to be a very scientific proceeding. The cause of the trouble is in the inner segment of the longitudinal arch, and there the deformity should be rectified.

OF THE FOURTH DEGREE.—Here the distortion is exaggerated to its utmost limit and the foot is fixed, feeling as if it were set in plaster of Paris. The decision has to be made as to between astragalectomy with free division of the resisting soft structures and amputation. The latter is very rarely called for, and many patients go on hobbling about with ulcerated feet.

Treatment of relapsed cases are best conducted on the lines advocated for the second and third degrees as above.

Congenital talipes calcaneus is seldom very resistant to treatment, and the same may be said of calcaneo-valgus. It is necessary to divide the contracted tendons and use the malleable iron splint for reposition, suitable manipulations being carried out twice daily.

CLUB-FOOT—ACQUIRED

Club-foot acquired—Causes.—The most usual cause is infantile paralysis, and less frequently spastic paralysis. Rarer causes are cicatrices such as result from burns; traumatism, such as injuries to bones and fractures, severance of tendons and nerves; inflammation, such as occurs in acute osteomyelitis when the rate of growth of one bone is arrested, while in the other it is normal; "talipes decubitus," a spurious form of talipes due to contraction of the tendo Achillis, and occurring in bed-ridden patients; and lastly hysterical talipes.

Talipes equinus.—The most common cause is spastic paralysis, less frequently infantile paralysis. The degrees of talipes equinus are as follows:—

THE FIRST DEGREE or right-angled contraction of the tendo Achillis. When the knee is fully extended the heel cannot be brought into complete apposition with the ground without pain or force. The results of this slight deformity are formation of corns beneath the heads of the metatarsal bones, slight lameness and shortening of the stride, and some inversion or eversion of the foot at the ankle. Many cases of so-called talipes varus or valgus are found to be equinus when carefully examined with the knee fully extended.

THE SECOND DEGREE.—The heel is raised well off the ground and progression takes place on the head of the metatarsal bones beneath which corns are found. The plantar fascia is frequently contracted in this degree.

THE THIRD DEGREE is an exaggerated condition of the second, and the foot is bent completely backwards. The morbid changes in the bones are such as would be expected from the more or less vertical position assumed by the foot. The plantar fascia and ligaments and posterior ligament of the ankle are contracted. So too are the tendo Achillis, the long flexors and the peroneus longus. In paralytic feet the extensors are fatty and degenerated.

The diagnosis is simple when the affection is well-marked and of the second and third degrees, but in the first degree or right-angled contraction of the tendo Achillis the affection is often overlooked. Talipes arcuatus and plantaris (pes cavus) are often due to paralysis of the interossei with some weakness of the long extensor tendons.

TREATMENT OF TALIPES EQUINUS.—In case of the first degree or right-angled contraction, manipulation and active and passive exercises designed to stretch the tendo Achillis may be employed. A toe-elevating spring attached to an outside vertical steel support is useful. But much time and trouble may frequently be saved by dividing the tendo Achillis, taking care, however, that the tendon does not become too long. The dorsi-flexion should be controlled by a "stop" at the ankle. In equinus of the second degree, section of the plantar fascia is called for, and when the sole of the foot is unfolded the tendo Achillis is divided. If the toes should be clawed, the extensor tendons may be divided opposite the heads of the metatarsal bones, at the same time that the plantar fascia is divided. Immediate after-treatment consists in the use of Scarpa's shoe or plaster of Paris; and to prevent the tendo Achillis becoming too long the ankle should be controlled by a "stop." Great attention must be paid to massage and the application of the induced current in paralytic cases. In the third degree the front part of the foot should be unfolded and the tendo Achillis divided subsequently. The wrench may be freely employed if the deformity is obstinate. Should it fail, astragalectomy is called for. In spastic cases the contracted tendo Achillis should always be divided despite the advice sometimes given to the contrary. By doing so, much lameness is avoided and the patient walks in comfort.

Acquired talipes calcaneus is the result either of infantile paralysis or is due to excessive lengthening of the tendo Achillis after an operation for talipes equinus. It is a very troublesome deformity to treat. Its aspect is entirely different from that of the congenital calcaneus. In the acquired form the heel is dropped and forms a very distinct prominence, and the arch of the foot is much increased, and there is some contraction of the plantar fascia. In all cases where the heel is much dropped and the arch of the foot unduly concave, especially if contraction of the plantar structures have ensued, the outlook is bad. The chief difficulty consists in keeping the heel up, and this is especially so when the cause is infantile paralysis.

Treatment.—Mechanically, a boot may be used having an outside steel support with a toe-depressing spring. Of course a contracted plantar fascia should previously have been divided. From an operative point of view many attempts have been made to shorten the tendo Achillis, but these as a rule are not successful because the tendon is extremely thin. The best results in the treatment of acquired calcaneus are obtained from tendon transplantation (*q.v.*).

Talipes calcaneo-valgus and calcaneo-varus are usually due to infantile paralysis. In calcaneo-valgus the heel is depressed and the foot is turned outwards, while in calcaneo-varus the foot is turned inwards. Sufficient indications for treatment are given under the headings of varus, valgus, and calcaneus.

Talipes Arcuatus and Plantaris or Pes Carus.—In these deformities there is increased concavity of the arch with a corresponding dorsal convexity. In talipes arcuatus the arch is increased, but the heel and the balls of the toes are in a horizontal plane. If the balls of the toes fall below the level of the heel, and the arch is at the same time increased, then the condition known as talipes plantaris is present. The causes are slight paralysis of the anterior muscles of the leg following dentition, measles, scarlet fever, chorea, infantile paralysis. They are also associated with Friedreich's disease, and some say (*e.g.* Duchenne) that they are due to paralysis of the inter-ossei and lumbricales. The most frequent symptoms are pain in walking, increase of the arch of the foot, and corns beneath the heads of the matatarsal bones. Treatment consists in division of the plantar fascia and the after-use of a Scarpa's shoe with a single uplifting movement in the sole.

Talipes Varus.—The acquired form is usually due to infantile paralysis and to one variety of progressive muscular atrophy in which the peronei muscles are the earliest to be affected. With regard to the appearances and treatment of a varoid foot they are sufficiently detailed in the description given of congenital talipes equino-varus.

Talipes valgus (acquired) arises either from infantile paralysis, spastic paralysis, rickets, or as a sequel to Potts' fracture. The appearances in treatment are precisely similar to that of ordinary flat-foot.

FLAT-FOOT

(SPURIOUS TALIPES VALGUS)

Definition.—Flat-foot is a deformity of the feet, often painful, characterised by abduction and eversion of the foot with loss of the arch.

Weak ankles or valgus ankles. Weakly children, especially those suffering from rickets, frequently turn their feet over in walking, and associated with this condition, which is due to relaxed ligaments, there is some genu valgum.

General Description and Appearance of Flat-foot.—All the changes of the foot are due to sinking of the so-called arch not only in the longitudinal but also in the transverse direction. The depression downwards and inwards of the head of the astragalus and the outward twist of the anterior part with extreme rotation of the foot are the immediate results. The foot is increased in length on the inner side and it is broadened. Flattening of the sole is also present, and the instep loses its roundness. The inner border is convex instead of concave, and is in contact with the ground. It is also thicker than normal. The heel appears to be shortened, the internal malleolus becomes extremely prominent in severe cases and descends downwards, inwards, and backwards, so that its tip is either in a line with or even behind that of the external malleolus. On the inner aspect of the medio-tarsal joint the head of the astragalus is prominent. It sinks downwards, forwards, and inwards, and the scaphoid becomes unduly prominent. The soft tissues over the astragalus and scaphoid are often hypertrophied, and false bursæ and thickened epidermis may be seen at these spots. In front of the medio-tarsal joint the inner border is sloped outwards, and the great toe is frequently valgoid. The outer border is commonly shortened and often raised from the ground. In many cases varicose veins and sweating of the feet co-exist; so that it is probable that flat-foot arises from defective innervation of the vessels of the legs and feet. In exceptional cases wasting of the tibialis anticus is seen.

Degrees of the Flat-foot.—For purposes of description there may be said to be four degrees. First degree or oncoming flat-foot.—There is noticeable some sinking of the arch when the patient stands and he is told to bear the weight fully on the foot. This sinking disappears on adduction of the foot, standing on tiptoe and on sitting. Pain is frequently present at this stage. Second degree or pronounced flat-foot.—The arch has sunk to some considerable extent although the head of the astragalus is not touching the ground. The deformity cannot be reduced by any effort of the patient, nor can the feet be voluntarily inverted. The peronei tendons, and the extensor communis digitorum are seen in relief, and there is considerable muscular spasm and pain. Third degree or spasmodic flat-foot. — The astragalus and scaphoid are touching the ground; the foot is very tender, and the patient can only hobble, and the deformity cannot be reduced either by the patient or by the surgeon. The peronei and extensor communis digitorum tendons are in strong relief, and the thickening of the soft tissues on the inner side is much in evidence. Fourth degree or osseous flat-foot.—In this degree the deformity is excessive, and marked changes occurring from arthritis are met with at the medio-tarsal and other joints. In some cases it is said ankylosis takes place.

The Etiology and Causation of Flat-foot.—In the majority of cases there are three factors involved, adolescence, feeble health, and strain on the feet out of proportion to the muscular development. Under the heading of feeble or impaired health contributing causes are, weakness following exanthemata, acute rheumatism, and anæmia. Excessive strain on the foot frequently arises from occupations involving long standing on weak feet (static flat-foot.) In addition to these general factors, local conditions are concerned, for example, bunions and corns on the outer side of the foot, hallux valgus, genu valgum, shortness of one limb, high heeled and narrow boots, gout, and injury.

Pathology and Symptoms.—Abduction of the foot is the position of weakness and adduction of strength and activity; for the usefulness of the foot varies with the preponderance of power of the adductor muscles. When this is lost weakness and pain ensue. In fact, flat-foot depends upon simultaneous relaxation of muscles and ligaments, probably due to vascular changes. The most marked change is in the inferior calcaneo-scaphoid and the calcaneo-astragaloid ligaments. As the ankle becomes more valgoid its ligaments suffer in proportion. The superficial part of the internal lateral ligament is elongated and thinned; the plantar fascia gives way, the muscles of the calf waste, and the calf loses its roundness, the peronei are in a state of tension and often stand out like cords. The alterations in the position of the bones can be readily understood if one grasps the fact that in flat-foot the astragalus descends and the bones in front of the medio-tarsal joint are twisted on their axes, so that the inner border of the foot is brought downwards and inwards. In many cases pain is present from the first, occasionally it is felt only after a twist or sprain of the foot. It commences as a feeling of fatigue succeeded by dull aching. This aching becomes more acute, and later assumes a sharp and intense form. The tender points in the flat-foot are well defined. They are found beneath the head of the astragalus and tuberosity of the scaphoid, also below and in front of the internal malleolus, on the dorsum of the foot and about the bases of the first and fifth metatarsal bone. The causation of the pain is first of all stretching of the muscles, ligaments, and fasciæ; and the acute pain is probably referable to surfaces of bone not normally in contact, being brought into relationship to one another at points which are not accustomed to pressure. Swelling of the feet and local puffiness are frequently seen over the tender points, and redness from time to time dependent in a degree on the amount of standing and walking. The existence of flattening of the sole is best ascertained by taking a tracing or outline of the "tread." Alteration in the gait: In cases of some severity the gait is lumbering and awkward; the patient is splay-footed; the foot is no longer elastic, and walking is further impeded by the attendent pain. In fact the patient is wooden-footed. Loss of shape in the feet is fully described under the sub-heading of appearances. Sweating of the feet is also mentioned above, and a probable explanation has been given. Flat-feet do not become cured without treatment. As a rule the pain and disability become steadily worse, and the patient has to seek relief from the inconvenience.

Diagnosis.—Probably there is no deformity so easily and so often overlooked as slight acquired valgus. The best method of detecting the trouble is to take a tracing of the sole of the foot. The writer has known flat-foot to be treated for rheumatism, gout, and for ostitis of the bones of the tarsus.

TREATMENT—*General.*—If anæmia be present, iron should be given for a considerable period. When the rheumatic taint exists, salicylate of soda in sub-acute cases, and in chronic iodide of potassium and tincture of gudiacum will be found serviceable. Gonorrhœal rheumatism is very intractable. In rachitic flat-foot cod liver oil, phosphate of iron, plenty of fresh milk and pure air, will go far to effect a cure. The relief of pain is often a pressing necessity. The surest therapeutic measure is rest, entire and absolute.

Local Treatment.—The measures we have at our command are rest, exercises passive and active, mechanical support, and operation. As to the treatment of the first and second degrees.—In static and rickety cases the first essential is absolute rest, and with this may be combined eversion of the foot. The patient should be told to sit on a comfortable sofa or bed "tailor-fashion."

The foot rapidly loses its spasm or pain, the arch rises and the deformity is temporarily relieved. When these occur, exercises should be carried out. Those most to be recommended are tip-toe movements. Their object is to strengthen the flexors of the toes, especially the long and short flexors of the great toe. These movements should be carried out in rhythm to the swing of a pendulum or to the beat of a metronome. After a few minutes' exercise twice a day, complete rest is advisable. Passive exercises may be carried out as follows:—The nurse takes the foot and performs a combination of extension movements at the ankle with rotation at the medio-tarsal joint inwards.

Supports.—In slight cases it is sufficient for the patient to wear a Thomas' boot. This consists of a wedging up of the inner edge of the sole and heel, so that it is one-fourth to one-third thicker at the inner than the outer edge. In very flaccid feet a valgus pad may be added. Numerous bandages are made, and many pads and surgical soles, but the vulcanised rubber valgus pad is the best. Whitman's brace is often of value. For this the foot should be corrected under an anæsthetic as much as possible, and a plaster cast taken of it, and the brace modelled on the plaster cast. Later on further correction should be done and another cast taken and the brace again modelled. In inveterate cases it is necessary to carry up the leg an outside steel support and to use a valgus pad in the boot, together with a T-strap to invert the foot. The treatment of the third degree of rigid or spasmodic flat-foot.—Before anything is done the patient should be placed entirely at rest for three to four weeks and the following measures may then be employed. Forcible rectification under an anæsthetic and retention of the foot in plaster, or the more gradual method by tenotomy, passive exercises, and the employment of a modified Scarpa's shoe. Forcible rectification is carried out either by the hand or by Thomas' wrench under an anæsthetic. The gradual method consists in the first place of rest, then division of the peronei and extensor communis digitorum, and occasionally of the tendo Achillis, of the application of a malleable iron splint, to be followed later by the use of a Scarpa's shoe. In all these cases it is essential to keep up massage of the muscles of the leg and the application of the constant current, and so soon as the foot can safely bear it, tip-toe exercises may be carried out. The treatment of the fourth degree.—It is in this degree, and when wrenching under an anæsthetic has failed, that operative interference on the bones is justifiable. The measures which have been carried out are resection of the sub-astragaloid joint, extirpation of the astragalus and of the scaphoid, transplantation of the posterior part of the os calcis and excision of a wedge from the head and neck of the astragalus. Of these six the first and last are in vogue. The first is known as Ogston's operation—resection of the astragalo-scaphoid joint. It is done under full antiseptic precautions, and with a chisel the cartilage and a thin layer of bone are removed from the astragalus and the scaphoid in such a way as to leave on the latter a concave surface. The bones are then pegged together with ivory, the wound closed, and the foot put up in a plaster case. Stokes' operation consists in the removal of a wedge of bone from the head and neck of the astragalus, fully adducting the foot, closing the wound, and putting it up in plaster. After all these operations great care must be taken to suitably support the foot so that no falling of the arch occurs. The writer has seen some recurrence after operation for want of this precaution.

Metatarsalgia

Definition.—This condition is a neuralgia, often extremely acute, chiefly situated in the anterior part of the foot. All authors agree that the immediate cause is pressure on the digital nerves at the head of the metatarsal bones. According to Morton, the pain is localised at the inter-space between the fourth and fifth metatarsal bones. But very often one finds that it has spread to the other digital spaces, although the pain is most severe in the neighbourhood of the fourth metatarsal bone.

Causation.—There are cases which show that either rheumatism or gout plays some share in the production of the disease, and in this way: these diseases cause a falling of the anterior transverse arch of the foot, and it is due to this falling that the nerves become pressed upon. The immediate incidence of the disease is often due to a blow or a strain in which the weight comes more immediately on the front part of the foot. In other cases the pain comes on after long standing or walking, especially in narrow boots. In most instances some degree of flat-foot is present, and this is an important point, as the flatness of the posterior part of the foot has spread to the anterior part, and the metatarsal heads have fallen out of position in such a way that pressure is caused upon the digital nerves. Mr. Robert Jones has shown that the pain in the neighbourhood of the fourth metatarsal bone is due to falling of its head and pressure upon the communicating branch between the internal and external plantar nerves which passes beneath the head of the fourth metatarsal bone. In other cases it seems that it is not this communicating branch which is pressed upon but the inter-digital nerves.

Symptoms.—The patient complains of either a dull aching pain or intense boring burning pain, beginning in the front part of the foot and radiating thence up to the leg and the thigh. The pain is such as to render movement impossible. It begins shortly after rising in the morning, and becomes worse before evening, unless the patient take his boot off and rest. In fact, a very frequent expression is that "they can get no rest until they remove their boot," and this they must do no matter where or under what circumstances they are. There is not, as a rule, redness, although in exceptional cases it is described as being present. If one feels carefully and takes a little trouble to press the metatarso-phalangeal articulations separately one finds that pressure, especially over the fourth metatarsal bone, readily elicits the pain. It can also be produced by squeezing the foot with the hand across the heads of the metatarsal bones. Frequently the affected foot is broader in that region than is normal, and on examining the sole one or two corns will be found, usually over the heads of the third and fourth metatarsal bones. These corns are indicative of the fact that those metatarsal heads have fallen out of their normal position. Frequently too, there is a peculiar twist in the foot; the portion in front of the tarso-metatarsal articulation is twisted inwards, so that the base of the fifth metatarsal bone is exposed to the pressure of the boot and the patient complains of constant pain at that spot.

Diagnosis. — Formerly this affection was confounded with gout or rheumatism, and vain efforts were made to treat it. It is sometimes mistaken for flat-floot: and as we have already mentioned, flat-foot is present in many of these cases. But it is not the flatness of the posterior part of the foot which gives rise to the acute pain. It is acute neuritis of the digital nerves.

As to *Prognosis,* in severe cases the only improvement or cure that can be effected is by operation.

Treatment.—In all instances it is advisable to search for evidence of rheumatism or rheumatoid arthritis and gout. In slight cases the acute attacks of pain may be relieved by removing the boot and soaking the foot in hot water; and the application of the oleates of morphia and atropine may temporarily relieve the pain. If boots designed on the following plan are tried they will be found useful. They should be narrow in the instep so as to compress the bases of the metatarsal bones, and where the heads of the metatarsal bones are there should be depressions hollowed out in the sole so that pressure is borne in walking not on the heads of the bones but behind them. Very often, however, one finds that this measure fails to give relief; there are then the following courses open: either to exsect the nerve or to amputate the toe or to remove the head of the metatarsal bone, around which the pain is greatest. As a rule this is the fourth, and it is astonishing to observe the good effects of this procedure. The pain immediately disappears never to return, and the mechanism of the foot is in no wise interfered with. In fact it is the one proceeding which gives permanent relief.

BOW-LEGS (CURVED TIBIA AND FIBULA)

Causes.—*Congenital curvature* of the legs is due to malposition *in utero.* It is usually associated with shortening of the limb and talipes equino-varus. The curve is usually anterior, and at the convexity of the curve there is frequently found a depression in the skin. This has been thought to indicate that compound fracture has occurred *in utero.* But this is not so. Treatment is generally of little value in these cases, but after childhood when the talipes has been corrected a wedge-shaped piece of bone may be removed from the tibia. Osteitis Deformans and osteo-malacia give rise to curvature in the tibia and fibula as in the other long bones.

Traumatic curvature of the bones arises most frequently from fracture. It is also met with after injury to epiphyses, either of the tibia or fibula. The growing power of one epiphysis is arrested, while that of the other remains unchecked, so that the longer bone is invariably bent. Such cases, however, are rather of the nature of surgical curiosities. *Syphilitic curvature* is interesting, and is more frequently a manifestation of the congenital form of disease than of the acquired. It should be carefully distinguished from *rickety curvature* by the following points. In the syphilitic form the curve is purely anterior, while in the rickety form it is antero-external or antero-internal. The syphilitic curve is usually situated at the middle of the shaft, while the rickety curve is more often in the lower third. The crest of the tibia in a syphilitic curve is smooth and rounded, and in a rickety curve sharp, while the surfaces of the tibia in the former are convex, and in the latter flat or concave. Syphilitic curvature of the tibia is best appreciated by looking at the bones from the side, when it is very striking. *Osteo-malacia and osteitis deformans* also cause curvature of the bones of the leg; the most common cause, however, is rickets.

Appearances.—In rickety bow-legs the tibia is flattened from side to side, and the curve is generally most marked at the lower third. The medullary canal is often narrowed in the middle of the shaft and enlarged at the extremities. On the concave side of the curve the bone is much thickened by sub-periosteal deposit, which acts as a supporting buttress to the arch. Curvature may take place in almost any direction, but the following types are found:—(*a*) An *external curvature* generally situated at the junction of the middle and lower third of the leg. (*b*) A more or less *anterior curvature* of the tibia occupying the whole length of the bone or only the upper or lower third. In these cases the heel is often raised, the foot pointed, and in walking is in a position of equino-valgus. (*c*) An *internal curvature* is present with flattening of the bones and the feet in a varoid position. Of these three types the first is common and the third rare. Occasionally there is seen a case with an internal curve in one leg and an external curve in the other.

Prognosis.—In bow-legs there is always a tendency to spontaneous rectification. This in slight cases is often complete, but in severe cases only partial. It is therefore unwise to allow any case to pass untreated, since, if the bones are soft, slight cases may very quickly become severe.

Treatment.—The method depends upon whether the bones are soft or eburnated, and upon the direction of the curve and the age and social status of the patient. If the bones are soft no operative measure is called for, and all forms of curvature except the marked anterior are amenable to mechanical treatment when the bones are soft. (1) *Constitutional treatment of rickets with local manipulation.*—This method is suitable for babies who have not yet walked, for children who are not weighty, and for those in whom the bones are not unduly soft and the curve is a general rather than a localised one. The manipulations are easily carried out by means of the nurse, and should be so done as to rectify the curvature. (2) *Constitutional treatment with mechanical support and manipulation* is adapted to the following cases:—When a curve originally slight is becoming marked; when a child is weighty and cannot be kept off his legs; when the curve is localised in one part of the bone more than another, and when the child is under four years of age and the bones are not hardened. It is not necessary to keep the child off its legs provided that the splints or mechanical apparatus are acting efficiently so as to control and diminish the size of the curve. The simplest form of apparatus is an inside wooden splint from the internal condyle to the internal malleolus for external curvature, and the reverse for an internal curvature. But when the curve is compound, *i.e.* when it is antero-lateral, a trough splint may be used with the angle of the trough placed posteriorly and internally so as to act as an opposing force to the antero-lateral curve. Should the curve be mainly anterior a more elaborate apparatus must be ordered.

OPERATIVE MEASURES.—These are osteoclasis either manually or instrumentally, linear osteotomy, and removal of a wedge from the bone. Operative interference is called for when the bones are so hard that mechanical treatment is out of the question; in children over four years of age; in cases of severe anterior curvature, and in marked instances of lateral curvature.

With regard to the choice of operation, the majority of surgeons prefer osteotomy, but some elect to perform osteoclasis. In young children in whom the bones are not very firm manual *osteoclasis* is to be preferred. It is carried out by fixing the thumbs at the summit of the curve and using them as a counter-resistance to the hands placed at the extremity of the curve; by a sudden combined movement of the two hands against the thumbs the bone is quickly snapped. Care must be taken that the bone is completely broken and not merely a greenstick fracture produced. Instrumental osteoclasis is carried out by one of the numerous osteoclasts. The advantage of osteoclasis is that no open wound is produced, and the fracture is simple. The leg is afterwards put up in splints or plaster of

Paris. *Osteotomy* is of two kinds, either the linear or the wedge-shaped. The limb may be put up into plaster of Paris. The removal of a wedge is rather more difficult, and much less satisfactory for the reason that it is often difficult to completely divide the periosteum posteriorly, but wedge-shaped osteotomy is called for when marked anterior curvature is present. The wedge is best removed by the chisel. Sometimes non-union occurs after these operations, and it happens more frequently than is suspected; but considering the enormous number of osteotomies that have been performed it is not a very serious danger.

Genu Valgum Varum and Recurvatum

Definition.—Genu valgum is a deformity of the lower extremity in which, if the legs are fully extended on the thighs, an angle obtuse externally exists at the knee-joint.

Varieties.—Rickety; static; rachitis adolescentium; traumatic, such as follows fracture of the lower end of the femur or separation of the epiphysis; inflammatory, due to ostitis about the lower end of the femur; and lastly, paralytic.

Causation.—Three causes are assigned—bending of the lower part of the shaft of the femur and upper part of the tibia, unequal growth of the epiphysial line, and relaxation of the joints. There can be no doubt that in rickety cases relaxation of the internal lateral ligament is the primary lesion, the internal condyle becomes prominent, and the other structures become adapted to the altered position of the limbs. The results are as follows:—Shambling gait, contraction of the biceps tendon and of the ilio-tibial band and external lateral ligament, rotation outward of the tibia, lateral mobility of the knee-joint, and some obliquity of the pelvis, and occasionally scoliosis. Flat-foot is very frequently seen in association with genu valgum.

Symptoms.—When the affection is coming on the patient complains of some difficulty in rapid progression, pain and tenderness over the internal lateral ligament, and becomes very readily tired.

Treatment.—In the rickety form the most important thing is to treat the constitutional trouble. Now in static genu valgum there are two stages met with, relaxation of ligaments and muscles, and osseous deformities arising as the result of relaxation. These stages afford us a guide in treatment. For neither in the softened stage of the bones in rickety genu valgum, nor in the early stage of relaxed muscles and ligaments in the static variety should an osteotomy, nor an osteoclasis, be performed. The means at our command therefore are general treatment, rest and local manipulation, mechanical treatment, and operative treatment. The general treatment is that for rickets. Manipulations are best carried out as follows:—The tibia should be brought firmly inwards with the right hand, while the left hand is held firmly against the lower part of the femur. The knee-joint must be maintained at full extension while these movements are carried out. Three or four movements inward are made, and the limb is allowed gradually to come back to its original position. It is important that the child should wear splints. The simplest splints are the long wooden outside splints, with a knee-cap fitted to them, and the splints secured round the pelvis. The more costly and more efficient arrangement consists of an outside steel support from the boot on both sides, and fixed around the waist by a steel pelvic band. Most cases recover in about nine months to a year under the combined effect of rest, change of air, good feeding, manipulation and splintings. But for those that do not, operative measures are called for.

These are osteotomy, osteoclasis, forcible manual rectification of the knee. Osteoclasis has been adopted of late, and appears to be likely to displace osteotomy. The object of osteoclasis is to fracture the femur just above the knee-joint, a somewhat difficult proceeding to carry out exactly. For this end various forms of osteoclasts or menches are used. Personally, the writer is not in favour of osteoclasis for genu valgum; he much prefers osteotomy, which is done either by Macewen's or Macormac's method. For practical purposes the latter method is best, and the writer is accustomed to use a saw in place of a chisel. Ogston's operation, or sawing off of the internal condyle, is by no means so good an operation as Macewen's or Macormac's, and in some cases distinct stiffness of the knee has followed sawing off of the internal condyle. After the operation the limb should be put into plaster of Paris for six weeks to two months, and subsequently it may be necessary to move the knee under an anæsthetic.

Genu varum.—Definition.—Genu varum is that condition of the legs in which a line drawn from the head of the femur to the middle of the ankle-joint falls inside the centre of the knee-joint.

Causation.—In the majority of cases rickets is the chief cause, and genu varum is in such instances constantly found associated with curved tibiæ. Indeed, the so-called genu varum is not limited to the knees. There is a general outward convexity of the femur and tibia, and as the knee happens to be situated very nearly in the mid-length of the limb, it is the most prominent part of the convexity. Genu varum is also met with after operation for genu valgum as the result of over-correction. It also arises from occupation, and is met with subsequently to excision of the knee. Genu varum is seen of all degrees from slight to very considerable deformity. The nature of the affection is evident at once on looking at the patient, and the treatment is conducted on the same lines as genu valgum. In cases in which the bones are soft, and the ligaments relaxed inside, splints with manipulation and massage are sufficient. When the bones are eburnated, osteotomy at the greatest point of curvature in the limb is necessary.

Genu Recurvatum

Definition.—A deformity characterised by hyper-extension of the knee-joint.

Occurrence.—It is seen associated with other conditions, namely, congenital and paralytic club-foot, rickets, deformities of one limb where an excessive strain has been put upon the sound limb; also in Charcot's disease, and as a primary condition in congenital displacement of the knee. If the deformity is a hindrance to progression, a walking apparatus with a flexion spring at the joint should be worn, or arthrodesis must be performed.

Coxa Vara

(incurvation of the neck of the femur)

Definition.—Coxa vara is a peculiar bending of some portion of the upper part of the femur in such a way that the head of the bone sinks downwards. This bending takes place in one of two positions, either at the neck, so that the head becomes horizontal, and is often twisted forwards, or the bend takes place just below the trochanter minor, so that the shaft forms here an obtuse angle.

Causation and Pathology.—The most usual cause is undoubtedly rickets, but there are other less well-known factors at work. Softening of the bone often occurs in adolescence, and has been ascribed to rachitis adolescentium, perhaps without sufficient reason. In boys who carry heavy weights, the neck of the femur sometimes gives. It is quite possible that some of the cases of coxa vara are due to slight inflammatory changes with softening at the epiphysial line. The affection is more common in males than females, and is more often unilateral than bilateral.

The neck of the femur gradually yields until the head of the bone is on a level with or below the top of the great trochanter. The neck is also bent in such a way as to form a curve with the convexity backwards. It is in this class of case that the difficulty in complete flexion of the thigh is met with. In a second variety of case, namely, bending outwards of the shaft just below the trochanter, this flexion difficulty is not encountered.

Symptoms.—In adolescence, without apparent cause, or following slight injury, the patient begins to limp and to complain of fatigue and pain about the affected joint on exertion. Shortening of the limb is soon apparent, and is accompanied by elevation of the trochanter above Nélaton's line. The limb is sometimes flexed to a few degrees, and is often rotated outwards. Abduction of the limb is also lessened. In fact, symptoms may be summed up as follows:—Peculiar stiffness of the hip, referred to "growing" pains; the stiffness is worse on rising after sitting for a time, but is relieved by complete rest; limping, if one side is affected; waddling, if both; shortening, amounting to as much as 1½ inch; prominence of the trochanters, especially on flexing the thighs; displacement of the trochanter above Nélaton's line, and backwards as well; rotation outwards of the limb, and eversion of the foot, limitation of inversion and final loss of abduction, with, in an extreme case, "scissor-legged" progression and inability to walk without crutches; tilting of the pelvis and consecutive scoliosis. The signs which are absent are:—Suppuration, thickening of the trochanter, tenderness on pressure, absence of the up-and-down movement on traction characteristic of congenital hip displacement.

Diagnosis.—It is very difficult to distinguish early coxa vara from coxitis, and the administration of an anæsthetic is often called for, but there can be no doubt that a small number of cases diagnosed as incipient coxitis prove to be coxa vara. Fracture of the neck of the femur, upper part of the shaft or separation of the epiphysis, especially if not seen until some time after the accident, may be difficult of identification. So too may congenital dislocation of a slight degree, but in the Röntgen rays we have a most valuable means at our command for diagnosis.

Prognosis.—Complete rest quickly relieves the pain, and the depression of the head and neck of the femur cease, and in some cases the length of the limb may be restored by traction. If the deformity is left to itself the patient can scarcely hobble along on account of the exceeding adduction of the limbs.

Treatment.—In the early stages entire rest, local massage, and passive motion in the direction of the limited movements will effect much. Failing these, complete recumbency and the employment of traction to the limb are of service. When the depression of the head has apparently reached its limit an oblique osteotomy through the great trochanter with the limb subsequently put up at an angle of abduction of about 25 or 30 degrees will do much to remedy the deformity. In those cases in which the bending is in the shaft just below the trochanter minor the wedge-shaped osteotomy is of service. The subsequent use of the cork sole will be necessary on account of the shortening if the affection is unilateral.

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Delirious Mania.—Synonyms.—*Acute delirious mania; delirium acutum; Bell's mania; typhomania.*

Definition.—An acute delirium characterised by precipitate onset, rapid course, fever, extreme prostration, and usually fatal issue.

History.—This disease was described fifty years ago by Dr. Luther Bell as "a form of disease resembling some advanced stages of mania and fever." Since then numerous cases have been reported in America, in this country, and on the continent. The term acute delirious mania has unfortunately been frequently misapplied to severe cases of mania or of excited melancholia, and in consequence some observers have been led to doubt the existence of Bell's mania as a distinct clinical entity. This would appear, however, to be a mistaken view, as there is abundant evidence of the existence of a quite sharply characterised acute delirious mania such as Bell described.

Etiology.—Most of the cases occur between the ages of thirty and fifty. The patients are frequently of neurotic constitution, and the symptoms may follow worry, over-work, or disappointment. Other cases have arisen in association with pneumonia, septic infection, insolation, alcoholism, and injury, especially of the head.

Symptoms.—There is usually, if not always, a prodromal stage during which the patient suffers from lassitude and depression of spirits, and often also from insomnia. The acute stage of the disease is entered on suddenly, and in the course of a few minutes the patient may pass into a state of the wildest excitement with vivid hallucinations, delusions of no fixed type, and outbreaks of great violence. He rushes about gesticulating, talking, singing, laughing, crying. He refuses food, and is entirely sleepless. This delirious stage may continue for several days, until at length he passes into a condition of extreme prostration. The movements become ataxic, and are interrupted by twitching and spasms. The talking is replaced by an unintelligible muttering. The tongue is now found to be covered with thick fur. Sordes appear upon the teeth. The pulse is quick and feeble. The temperature is found to be from four to six degrees above the normal. The whole aspect of the patient is that of profound nervous exhaustion. The appearance of this typhoid stage is very characteristic, and suggested the name typhomania originally applied to the disease by Bell.

The early excitement, the want of food, and the want of sleep result in rapid wasting of the body, which may become quite extreme under the drain of a colliquative diarrhœa which sometimes sets in. Perspiration is often profuse, and eruptions, pustular, bullous, or petechial may be present. The urine is scanty, high coloured, and may be albuminous. Gradually the patient sinks into deep coma, and death takes place in from three days to three weeks from the onset of the delirium. In the rare cases that recover the patient does not become comatose, but passes through a prolonged convalescence, regaining his bodily and mental health very slowly, and often imperfectly.

Prognosis.—As already stated, the disease is usually fatal, but the mortality is variously stated by different writers. When death does not occur a good deal of mental feebleness, amounting even to complete dementia, may be left behind.

Pathology.—The principal anatomical changes are congestion of the meninges and of the cerebral cortex, and occasionally areas of softening. Punctate hæmorrhages are present, and leucocytes and red blood-corpuscles are found in the perivascular spaces.

The disease appears to be an acute infection. Organisms have been isolated from fatal cases. Rasori, for example, obtained from an area of softening in a fatal case pure cultures of a small bacillus with rounded ends which grew in the ordinary culture media at the temperature of the body, or of the room, and which was stained by the ordinary aniline dyes, but not by Gram's method. Rabbits inoculated beneath the dura died in two days; beneath the skin in four to six days; and in both cases cerebral congestion and œdema were found.

Diagnosis.—The clinical history of Bell's mania differs considerably from that of ordinary acute mania or excited melancholia, though some difficulty may be experienced in diagnosis during the period of excitement. The precipitate onset, the extraordinary intensity of the symptoms, the rapid changes in the *type* of the delusions and hallucinations, and the fever are points of importance. Greater difficulty in diagnosis is sometimes presented by other febrile diseases associated with delirium. The delirious stage of Bell's mania is sometimes of very brief duration, and the patient quickly passes into the condition of prostration, to which the term "typhoid" has been applied. In this condition the case is readily mistaken for one of typhoid fever, but a consideration of the history, examination of the patient, and the use of Widal's test, should lead to a correct diagnosis. Acute pneumonia with delirium, and uræmia with maniacal symptoms, may give rise to difficulty in diagnosis. The disease can hardly be mistaken for delirium tremens. A febrile delirium occasionally occurs in association with malarial poisoning.

Treatment.—The treatment of this disease cannot readily be carried out in a private house, as the patient during the early stage is excessively noisy and often violent. The patient is best kept by himself in a large, cool, darkened room, and nursed by experienced attendants. He should be kept in bed, his movements being limited by the use of a restraining sheet. Forced feeding with concentrated fluid nourishment at short intervals is essential. Some writers recommend the administration of a calomel purge at the outset. None of the hypnotic drugs seem to give very satisfactory results. Opium is considered dangerous. Hyoscine is sometimes useful in limiting the excessive muscular activity, but must be used with great caution. Several writers strongly recommend the use of ergotin. Alcohol should not be used in the earlier stages, but may prove of great value when the pulse is failing.

In cases where recovery is taking place, careful nursing for a prolonged period is necessary, and the patient must be guarded very carefully from all excitement and fatigue, such as is entailed by too early visits from friends. After recovery the patient should, if possible, go to live quietly in the country or at the sea-side, and refrain entirely from mental work for at least a year.

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Delirium tremens.—Delirium tremens (*mania a potu*) is essentially an insanity of short duration, occurring in the course of chronic alcoholism, and due not to the direct action of alcohol on the nervous system, but to nutritive changes brought about by usually long-continued alcoholic abuse. It is characterised not only by temporary mental alienation, but by disturbances at a lower level indicated by motor and sensory symptoms.

Symptoms.—The onset of the attack is frequently determined by a temporary excess, or may be precipitated by the occurrence of an accident or the supervention of some acute illness such as pneumonia. The patient will almost always be found to be a confirmed tippler, although it not infrequently happens that he has seldom or never been actually drunk.

At the outset of the attack the patient often complains of sleeplessness at night. He suffers from great distaste for food, and often for a few days for drink also. His movements are noticed to be markedly tremulous. In conversation he is apt to be somewhat incoherent and his attention is difficult to fix. The acuter symptoms frequently come on at night. He becomes extremely restless, and is the subject of a busy delirium in which he wanders about the room moving the chairs or arranging the bedclothes, talking constantly the while. He is very suspicious, and frequently looks behind the curtains or under the bed to see if anything likely to hurt him is concealed there. Visual hallucinations appear, usually of a terrifying nature, whence the disease has acquired its popular name of the "horrors." The terror inspired may be so great as to lead him to jump out of the window or run into the street half naked. Auditory hallucinations are less constant, but are not rare.

The tongue is moist, tremulous, and covered with a thick whitish-yellow fur. The pulse is rapid and soft. The temperature is usually moderately raised.

After a few days, in a mild case, the symptoms gradually subside, and sleep and appetite return. The tremor persists for a few days longer. In severe cases the course of the disease is more protracted, the insomnia persists, the distaste for food is aggravated and may be associated with troublesome vomiting, exhaustion becomes extreme, and death may occur from heart failure. In other cases again the acute symptoms pass off, but the patient remains for a time the subject of hallucinations, or of insanity with melancholic delusions. After repeated attacks the brain becomes more and more enfeebled until a condition of dementia is reached.

Prognosis.—In a strong patient with a healthy constitution recovery is the rule. In all cases the strength of the patient, his history as to intemperance and previous attacks, and the severity of the attack, have to be taken into account. An attack in which the patient imagines he sees small objects such as spiders or beetles about the bed is apt, other things being equal, to be more grave than one in which he sees larger objects such as cats and dogs. Where no history of long-continued intemperance can be obtained, the presence of peripheral neuritis, indicated by extreme tenderness of the calves on deep pressure, will tell its own tale. All patients must be examined carefully for evidence of acute disease, particularly pneumonia, the symptoms of which are sometimes latent.

TREATMENT.—Delirium tremens being a self-limited disease resulting often in extreme exhaustion, the principal indications for treatment are to maintain the strength and to procure sleep. The former is best carried out by keeping the patient in bed, and administering easily assimilable nourishment—milk, strong soups, etc.—at short intervals. Every effort should be made to keep the patient in bed by persuasion, but in cases with very great excitement and violence it is better to resort to mechanical restraint than to permit prolonged struggling between the patient and his attendants.

As to procuring sleep, it has to be remembered that in a mild case sleep will tend to come naturally in the course of three or four nights, and it is not advisable to push the use of hypnotics too early. Nevertheless in the majority of cases the early excitement can be quickly reduced, the restlessness and consequent muscular exhaustion diminished, and the coming of sleep hastened by the judicious use of hypnotic drugs, although it cannot be claimed for them that they cut short the disease. As to the drugs to be used, a combination of chloral and bromide of soda judiciously administered and with due regard to the effect obtained will be found perfectly satisfactory in most cases. Opium was at one time far more used than at present. Most of the newer hypnotics have been extensively tried during recent years.

Alcohol should be avoided as much as possible in the treatment of delirium tremens. Still it is of value in a few severe cases with marked prostration, and especially in such cases when complicated with pneumonia.

After recovery from the immediate attack further treatment is indicated on the lines laid down in the article "Alcoholism."

Delusional Insanity. *See* INSANITY.

Dementia. *See* INSANITY.

Dengue.

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SYNONYMS.—*Dandy, Breakbone, Three days' fever, Abu rokab, Kidinga pepo, Scarlatina rheumatica, etc.*

DEFINITION.—A specific disease, severe but not dangerous; usually distinguished by fever of sudden onset, intense muscular and joint pains, and later a rubeoloid eruption. Endemic in some countries, it usually occurs in regional epidemics, and since originally recognised (1779) has thrice assumed pandemic proportions.

GEOGRAPHICAL DISTRIBUTION.—Among Eastern countries, Arabia, China, and particularly India and the island countries adjoining, are subject to its visitations.

Epidemics in limited parts have occurred on all sides of Africa, except the extreme south, and in Egypt and Zanzibar especially.

In Europe its outbreaks have been confined to Spain, Greece, and Asia Minor.

In the Western Hemisphere the list includes Bermuda, the West Indies, Southern States, parts of South America, and even as far south as Tahiti. It has also reached Sydney and Brisbane in Australia.

Sporadic cases are reported by Sandwith in Egypt, and also occur in Arabia, Zanzibar, Bermuda, West and East Indies, and Honduras. In some of these the disease was apparently first introduced during an epidemic period.

ETIOLOGY.—In suddenness, rapid diffusion, and the enormous proportion (sometimes as great as four-fifths) attacked in a community, dengue has no equal except influenza. Again, in dependence on temperature and altitude it resembles yellow fever, which in some places it has closely preceded or followed, in the latter event reproducing, according to Smart, the features of the milder cases.

Essentially a disease of the tropics, and usually selecting there the hot season, its ordinary limits are given by Manson as 32·47 N. and 23·23 S.; yet in warm weather epidemics, always checked by winter, have extended to Southern Europe and Philadelphia.

Coast and river districts and low levels suffer far more severely than inland parts and high altitudes, perhaps a result of freer communication, a possibility supported by the fact that towns, particularly the dirty overcrowded parts, usually provide the starting-point and chief field for its outbreaks. Additional evidence that it is communicable is found in the numberless instances recorded of direct conveyance of infection, the particular liability of immediate attendants on the sick, and in its advance along the trade routes.

All ages and both sexes, too, seem equally susceptible, and although coloured people have occasionally suffered least, they evidently possess no immunity.

Although no organism has yet been definitely proved to be the cause, M'Laughlin found in the blood of twenty patients, without exception, a micrococcus possessing unique biological characteristics, and succeeded in cultivating it in media and in blood; he was, however, unable to investigate further.

Whatever be the value of his researches, the striking resemblance in mode of spread which the foregoing facts show that it bears to other disease of proved parasitic origin strongly suggest a like cause—an organism dependent for its highest activity upon high temperature, low levels, lack of air-space, dirt, and overcrowding. One cannot doubt its being communicable, but its occurrence in epidemic form may depend upon these other factors, a supposition favoured by the immunity enjoyed in 1871-72 by ships of the Indian Squadron in free communication with infected ports, and by the fact that, although actually introduced into some, only isolated cases occurred. Together with the short incubation (a few hours to five days), it would largely explain the chief arguments against its infectious nature, namely, the large proportion almost simultaneously attacked, dependence on local conditions, and presence of sporadic cases before epidemics.

CLINICAL FEATURES.—The patient is often attacked with absolute suddenness, and almost at once prostrated, but sometimes premonitions, such as malaise, painful twinges, gastric uneasiness, thirst, headache, or lassitude, precede the onset by some hours; more frequently, in Maclean's opinion, towards the end of an outbreak.

Headache, severe, perhaps paroxysmal, and associated with a sense of fulness in the skull, rapidly increasing fever, and usually severe pains, usher in the attack. The first intimation is not infrequently given by twinges in a finger or elsewhere, thence rapidly extending to other parts of the body.

Pain and sense of fulness in the eyes, which become injected and ferrety, aching in the loins, and great susceptibility to external air, though rarely definite chills or rigors, are also complained of. The skin is hot, with occasional fugitive perspirations, and the face suffused with a deep flush, disappearing momentarily on pressure, and often attended by puffy swelling. This, which is not invariable, and is usually transient, constitutes the initial eruption, and may extend over part or most of the body. With it the buccal mucous membrane is frequently involved, reddened, congested, or even superficially ulcerated, resulting in soreness of mouth and throat. Throughout the entire illness anorexia is a prominent symptom, while gastric uneasiness, nausea, or vomiting are often present, especially after ingestion of unsuitable food.

The tongue is coated or clean, with red edges and enlarged papillæ, the bowels are usually confined and the urine is scanty and high coloured, though rarely albuminous.

The pulse is rapid or little affected, and the respirations are hurried from pain and fever.

The temperature rapidly rises, perhaps as high as 103° to 105° F. or more, but declines quickly; to find it maintained above 102° being in Fayrer's experience exceptional. Previous disease, such as malaria, may modify this, like other symptoms, by increasing its severity and persistence and making it more ague-like.

By giddiness, pain, and faintness the patient is soon completely prostrated, and his sufferings are increased by his extreme restlessness. No posture is comfortable, while every movement causes increased torment.

Marked nervous symptoms are rare, but convulsions occasionally mark the onset in children, and at the extremes of life resolution may be attended by great depression. Insomnia, too, is a common feature; delirium may accompany high fever, and temporary loss of smell or taste has at times been noted.

The pains experienced may be intermittent in character and referred to muscles, bones, and joints. There is often an uneasy stiffness, unaffected by passive movement, but acutely painful on any muscular contraction, worse too after rest, and frequently combined with a sense of powerlessness. There may be neuralgic, dull, aching, or boring pain, most noticeable in the morning, sometimes metastatic, and possibly accompanied by swelling of joints.

Second Stage.—This first stage terminates after one to three days, gradually, or more often by crisis, marked by profuse sweating and diuresis. Bilious diarrhœa and hæmorrhages may also occur now or earlier, particularly epistaxis, which has a great effect on the headache. With fall or remission of temperature the pains abate and the patient feels well, though debilitated and perhaps subject to "reminders," such as twinges, anorexia, languor, and irritability.

Third Stage.—From the fourth to the eighth day of illness the terminal eruption appears, sometimes attended by slight transient return of fever, and the pains recur with, at times, greater intensity than before. This eruption is extremely variable and may be scarlatinal, rubeoloid, or urticarial; it is usually present to some extent. Tingling, itching, and numbness of the fingers often foreshadow its appearance on the palms—its frequent starting-point. It may begin there as small red spots, disappearing on pressure, which gradually coalesce and perhaps spread all over the body; in some cases combined with swelling.

Though generally most profuse on hands and knees, the face may be first affected, as in Christie's cases; it fades in order of appearance. A branny desquamation, often trifling, practically always follows it. This begins in about three days, and may persist for several; it is accompanied in many cases by intolerable itching.

The eruption usually disappears in twenty-four hours, though it may persist two or three days, and fever, if present, is likewise transient and very slight.

At the commencement of this stage, swelling of the cervical, inguinal, and axillary glands not uncommonly occurs, if it has not done so before. At Zanzibar the occipital glands were always involved. The already noted buccal congestion and swelling of joints may also make their appearance now, and rarely, ptyalism, orchitis, or angina.

Varieties occur in separate outbreaks and different localities, the cases differing in severity and in the period of incidence, relative prominence, or even existence of certain features, such as swellings of joints and glands, pains, hæmorrhages, eruptions, or the occurrence of relapses.

For instance, in Calcutta, in 1853, the characteristic pains were infrequent, and the buccal mucous membrane was prominently involved.

In mild cases, too, the course may be very short and all symptoms absent, except perhaps slight fever and eruption.

True relapses occur, sometimes due to indiscretions in diet or exposure.

Serious complications, excepting rare hyperpyrexia and mild inflammation of serous membranes, are hardly ever seen unless from previous disease.

Sequelæ.—With desquamation the pains may vanish, but in many cases they persist in one or more joints and adjacent tissues, or perhaps return after an interval of days or weeks. They often trouble the patient for a considerable time, possess many of the features seen in the acute stage, and are specially liable to occur in elderly people. The shoulders, wrists, knees, and feet are the parts more commonly involved.

Some debility, with anæmia and anorexia, is usual after severe attacks, and insomnia, swollen glands, and furunculosis are not infrequent sequelæ.

DIAGNOSIS is only difficult in sporadic cases or in the presence of concurrent epidemics of influenza or yellow fever. Hamilton West demonstrates its resemblance to mild attacks of the latter, which he considers, however, distinguished by the different course, the greater frequency of jaundice, albuminuria, and hæmorrhage, and absence of the characteristic eruption.

From influenza its distinction, at times a difficult task, is aided by infrequency of complications, the season of year, and the pains, eruption, and desquamation.

Some of these features, together with epidemic prevalence, distinguish it from rheumatism, and, aided by geographical distribution, from measles and scarlatina, while in malarious countries blood examination is valuable for doubtful sporadic cases.

PROGNOSIS is always favourable, unless in patients already greatly debilitated, but by its effects dengue may predispose to other illness. Charles describes a rare pernicious type, with œdema of lungs, cyanosis, coma, and hyperpyrexia, as occurring in Calcutta.

TREATMENT.—As the disease must run its course, this must be directed towards (1) limiting its diffusion, (2) avoidance of complications and sequelæ, and (3) relief of symptoms.

As complete isolation as possible in clean well-warmed and ventilated rooms, easily digestible nutritious diet, a simple diaphoretic, rest and warmth for all stages will best attain these ends, but can rarely in this disease be thoroughly carried out. Purgatives and emetics should, from the distressing movement they occasion, be given only if specially indicated.

For high temperature, sponging, or in the rare event of hyperpyrexia, ice and quinine hypodermically should be made use of, while gastric irritability is allayed by ice to suck, effervescents, and the usual remedies. Quinine is indicated in cases complicated by malaria. An ice-cap and mustard pediluvia are of value in relieving headache, or antipyrin and phenacetin may be given. These drugs will also relieve the rheumatic pains, others recommended for the purpose being belladonna and salophen, while locally small sinapisms and liniments containing chloroform may be applied. If very obstinate, however, opium must be prescribed.

Warm baths and camphorated oil help to allay itching, while debility and anæmia demand the administration of stimulants, iron, and tonics. For the rheumatic sequelæ, tonics, salicylates, and potassium iodide, with massage and faradism, have been advocated. Complete change of climate may be necessary.

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Dentition. *See* Child, Development of—Teeth.

Dermatitis Herpetiformis.

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Synonyms.—Hydroa, Duhring's disease, pemphigus pruriginosus, dermatitis multiformis, including herpes gestationis. The alternative name, Hydroa, appears to the writer to present obvious advantages over the cumbersome title applied to this disease by Duhring, which is, however, here employed in accordance with general usage.

Dermatitis herpetiformis includes a large and somewhat inchoate group of vesicular and bullous skin eruptions, having close relationships on the one hand with pemphigus, and on the other with the bullous erythemata. It is only within the last fifteen years that the disease, as now understood, has been generally recognised as a separate morbid entity. Hebra certainly observed cases, which he regarded as variants of pemphigus, and Kaposi maintains this view even to the present day. Bazin dimly foreshadowed the outlines of the group, while a posthumous paper by Tilbury Fox, published in the *American Archives of Dermatology*, clearly showed that that distinguished author distinctly recognised the special clinical features which characterise the disease, many typical examples of which he had accurately observed. It is, however, to Duhring that the credit is usually ascribed of finally isolating dermatitis herpetiformis from its congeners, and his admirable description, published in 1884, still holds good to a large extent and almost justifies the patronymic nomenclature frequently applied to the disease. More recently Brocq, of Paris, has published many elaborate articles on the subject, insisting especially upon the existence of two separate forms of dermatitis herpetiformis—the acute and subacute,—but these distinctions are not admitted by Duhring. Unna and many other writers have combined to create a mass of ill-digested literature, out of all proportion to the practical importance of the subject, dwelling mainly upon petty details of little scientific value, as our knowledge of the etiology of the disease—which must always be the rational basis for scientific classification and differentiation—remains virtually an unknown thing.

There is much difference of opinion as to what "types" ought to be included under dermatitis herpetiformis, more especially as to the following:—(1) hydroa (vel herpes) gestationis, (2) impetigo herpetiformis, (3) hydroa vacciniforme, (4) Hallopeau's chronic pustular dermatitis in excentrically progressive groups. Without entering into any elaborate discussion on the point, it may be said that dermatologists are now practically unanimous in accepting the first and in rejecting the three last as forms of the disease under consideration. Unna and Jamieson are strongly opposed to the inclusion of any form of skin disease which is not essentially chronic in its course, and in this view the writer fully agrees.

The "types" described by Duhring are generally accepted, and are as follows:—(*a*) The *erythematous,* including the *urticarial* forms; (*b*) the *vesicular,* or commonest forms; (*c*) the *bullous;* (*d*) the *papular,* seldom extensive; (*e*) the *pustular,* in which pustules are present from the first, and which many would classify along with impetigo herpetiformis; and (*f*) the *multiform,* in which the several foregoing types co-exist. With reference to type (*e*), Allan Jamieson has recorded a case in the *International Atlas of Rare Skin Diseases* which presented features common to impetigo herpetiformis and dermatitis herpetiformis, forming, as he thinks, a connecting link between the two. The former disease being now regarded as not essentially connected with pregnancy or the puerperal state, Jamieson thinks that its inclusion under dermatitis herpetiformis would tend to simplify our conceptions.

Dermatitis herpetiformis is essentially a chronic affection extending over several months or years, with relapses and recurrences at varying intervals, the skin inflammation being a superficial one, and the lesions showing a marked tendency to "herpetiform" grouping, *i.e.* for various vesicles to form closely aggregated on a common erythematous base. In different attacks the type of the disease may vary. Itching, burning, or pain is almost invariably present, the first being the commonest form of dysæsthesia, and often of intolerable intensity. All are agreed that the disease is essentially of neurotic origin, and occurs in persons of nervous temperament, while a certain proportion of its subjects become insane. Oddly enough, the general health in mild cases, or during the early stages, is often practically unimpaired.

Symptoms.—The onset of dermatitis herpetiformis is often preceded for several days by loss of appetite and malaise, sensations of chilliness or flushing, and constipation. The temperature may be raised a degree or two before the cutaneous eruption appears, and there is often considerable preliminary itching *sine lesione.* After the eruption has appeared these symptoms usually increase in intensity, and serious constitutional symptoms may occur, especially in the bullous and pustular forms. Such cases are, however, certainly infrequent, the general health being usually but very little affected. The rash is always bilateral, and usually fairly accurately symmetrical; it may occur on any part of the body, but is most frequently observed on the flexor surfaces of the arms and wrists, on the abdomen and outer sides of the thighs. The lesions at first consist of rose-red erythematous patches or slightly-raised papules; these rapidly coalesce to form plaques, which are often circular and average about half an inch in diameter. These circular patches or plaques present a raised red margin, and their centre soon becomes flattened, depressed, and of purplish colour, the appearance closely resembling that of the lesions of erythema circinatum. After a day or two vesicles or bullæ form on the spreading margin, some cases thus simulating a herpes iris (Hallopeau); the vesicles vary in size from a millet seed to a pea, or may even become as large as an inch in diameter. In rare cases bullæ may develop in the centre of the patches, and both bullæ and vesicles may form independently of erythema. On the other hand, the erythematous patches do not necessarily vesicate or bullate.

While the eruption is developing very marked pruritus is almost always present, and ephemeral urticarial wheals sometimes manifest themselves. As the vesicles continue to appear, the itching is supplanted by feelings of burning, pricking, or actual pain, only relieved by the escape of the fluid contents of the lesions; this evacuation does not, as a rule, occur spontaneously, but as the result of the rubbing or scratching of the patient. The vesicular contents are usually clear and serous, but may become purulent. Contiguous vesicles tend to coalesce, forming irregular, multilocular blebs, which assume a withered, puckered appearance as they begin to disappear. Finally the erythematous base upon which the blebs originally formed may disappear, the surrounding skin showing no sign of inflammation. When the vesicles are small, and the erythematous base persists, the resemblance to herpes zoster is considerable, and many cases reported as bilateral or universal zoster are undoubtedly of this nature.

Appearances such as have been described occur in successive crops either in rapid succession or at intervals of several weeks, but in any given case lesions of various ages (wheals, erythema, papules, vesicles, bullæ, and scales or scabs) are always present simultaneously during an attack, although one or other elemental lesion usually predominates. As a rule itching is more severe in the vesicular and bullous types than in the other varieties of the disease, and in the vesicular cases the intervals of quiescence between the attacks are usually short, the duration of the disease generally very protracted. In the majority of cases only some macular pigmentation remains after the subsidence of the eruption; but in exceptional instances, observed by the writer, minute, shallow atrophic pits, herpetiform in arrangement, have remained as evidences of former attacks, facilitating diagnosis in some dubious cases.

The *bullous* type is signalised by the suddenness of development of round or irregular tense blebs with very little erythema; but minute vesicles or pustules and a few papules are generally also present. When the blebs burst or are broken they dry up, forming yellowish scabs or crusts, which drop off, leaving pigmented patches. If blebs appear in rapidly successive crops, considerable infiltration of the skin may ensue and subjective symptoms may be very troublesome. The general condition may be unaffected, but there is generally a certain amount of pyrexia with chilliness and furred tongue.

The *multiform* type is characterised by the coexistence of the several types already described, and is undoubtedly the most frequent and typical form of dermatitis herpetiformis. No further description of it seems necessary here, but a word must be said regarding Hydroa (vel herpes) gestationis, which by universal consent is now regarded as a form of dermatitis herpetiformis occurring either during pregnancy or the puerperium. It seldom appears before the fourth month of pregnancy, but after that date manifests itself with gradually increasing frequency up till parturition. Fresh relapses often occur after delivery and throughout the puerperal period. In some cases seen by the writer the disease first appeared two or three days after delivery. A woman who has once had an attack of hydroa as the result of pregnancy almost always has fresh attacks, and usually of increasing intensity and of earlier occurrence, with each succeeding pregnancy. Subsequently relapses usually occur independently of pregnancy, and the case becomes identical in every way with ordinary dermatitis herpetiformis. The writer has observed one case of hydroa gestationis recurring in seven successive pregnancies; the bullous affection then became practically continuous and universal; the conjunctivæ were involved, then the mouth and œsophagus were attacked, and finally the patient died after twelve years' illness from the perforation of ulcers in the ileum, presumably of the same nature as the skin lesions.

The involvement of mucous membrane in dermatitis herpetiformis is, however, of very exceptional occurrence, in marked contrast with what obtained in the bullous erythemata.

ETIOLOGY.—That the disease is a neurosis is indicated by the severity of the disordered sensations preceding and accompanying it, by its peculiar paroxysmal course, and by the coexistence of other well-marked nervous symptoms in many of the cases. It manifests itself frequently after some mental shock or emotion, worry, or nervous breakdown, but a considerable proportion of cases have been recorded in young adults otherwise in good health. Its occurrence as the result of pregnancy suggests its "reflex" origin, but beyond this our knowledge of the etiology of hydroa is as purely hypothetical as that of pemphigus and other bullous diseases. No relationship with gout or renal disease has been established.

MORBID ANATOMY AND PATHOLOGY.—In sections from a case of Jamieson's of the erythemato-vesicular type there was evidence of the origin of the vesicles in the upper papillary layer, although fully formed vesicles of a loculated character were also met with in the epidermis in close relationship to the hair follicles. Elliot found some association between the sweat ducts and vesicle formation, and located the commencement of the process in the rete and interpapillary spaces. Gilchrist, however, believes that the upper layer of the corium is the starting-point, and has found no alteration in the cells bordering on the sweat ducts. Both observers found small cell infiltration in the corium, which in Gilchrist's case was found to consist largely of eosinophile cells, and these had also penetrated to a certain extent into the epidermis. Leredde and Perrin, who first demonstrated the abundance of eosinophile cells in the serum of the bullæ and in the blood in dermatitis herpetiformis, consider it as diagnostic and as differentiating the disease from pemphigus; this has been disputed by Neusser and many other observers, who have found excess of eosinophile cells both in the blood and serum of vesicles of eczema and pemphigus. The normal proportion of these cells is from 1 to 2 per cent of the leucocytes. In a case of Morris and Whitfield's the eosinophilia rose from 4·9 per cent of all leucocytes present in the blood at the commencement of the attack to 12 per cent when the eruption was at its height. Whitfield found from 8 to 15 per cent of eosinophiles in the blood of another case of dermatitis herpetiformis, and double this amount in one of hydroa gestationis. In a case reported by Danlos eosinophiles were present to the amount of 25 per cent in the blood and 54 per cent in the serum of the blebs. On the other hand, Drysdale showed at the Pathological Society of London specimens from the blood of a case of true pemphigus containing 60 per cent of eosinophiles, and in which the serum of the blebs was extremely rich in eosinophiles; while Peter contends that eosinophilia is a more marked feature of eczema than of any of the bullous disorders which might be classified together as pemphigus.

The occasional occurrence of associated recurrent hæmo-porphyrinuria, as recorded by M'Call Anderson, indicates that the underlying neurosis is probably of a paroxysmal nature.

DIFFERENTIAL DIAGNOSIS.—Many authorities assert that it is impossible to diagnose a first attack of dermatitis herpetiformis with positiveness, and that a firm diagnosis can only be established after watching a certain number of recurrences. This view is not endorsed by the writer's experience, although the value of the history of such cases is always very great. The similarity of the lesions to those of pemphigus and erythema multiforme may easily give rise to errors in diagnosis, especially as a history of repeated recurrences is common to all three, although in different degrees. The blebs of pemphigus characteristically arise upon skin which shows no sign of erythema and are not intermingled with the scattered papules, vesicles, and pustules so frequently present in dermatitis herpetiformis; they are usually large and show no tendency to herpetiform grouping, while the accompanying constitutional symptoms are generally severe. Itching is a marked or even predominant feature of the disease under discussion, but is seldom present in pemphigus. The therapeutic test is undoubtedly of considerable value, as most cases of true pemphigus yield rapidly to arsenic, which has no beneficial effect in dermatitis herpetiformis. The alleged value of eosinophilia as a diagnostic point has already been referred to.

Erythema multiforme is an acute disease running a rapid course, and usually with characteristic localisation, often also involving the buccal mucous membrane. Erythema predominates over vesication, and the lesions never exhibit herpetiform grouping.

The Hydroa vaccinoforme of Bazin (Hutchinson's summer prurigo) occurs in young persons, and only in spring and summer; its lesions are confined to the face and other parts exposed to sunlight, and tend to diminish with advancing age; they leave indelible pitted scars.

Impetigo herpetiformis presents many striking points of distinction. The pustules of which the eruption is composed are minute, miliary; they form little groups which spread centrifugally; itching is generally absent; the general condition is grave from the start, and death generally ensues in a few days or weeks.

Vesicating urticaria may also give rise to some doubt as to diagnosis; but it almost always occurs in children, and the concomitant phenomena readily distinguish it from dermatitis herpetiformis.

Hallopeau's chronic pustular dermatitis in excentrically progressive groups is a very rare disease, all the recorded cases having been observed in St. Louis Hospital, Paris. It appears to be a local pus inoculation of intense severity leading to deep involvement of the skin, which becomes much thickened and fungates. A case recorded by Wickham seems to show that this condition may supervene as an epiphenomen in cases of Duhring's disease.

The points of differentiation from herpes or any form of "eczema" have been sufficiently dwelt upon in the preceding portions of this article.

Prognosis. — Although the general health may in many cases of dermatitis herpetiformis be but little affected, the favourable prognosis attached to the disease, and considered by Duhring as one of its salient features, is far from being entirely justified; the records of fatal cases are now by no means rare. The disease, as a rule, ceases spontaneously rather than as the result of treatment, after lasting for many months or years; or a fatal result may ensue from exhaustion or septicæmia. Generally speaking, the erythematous forms and those connected with pregnancy are the most benign, while in the bullous and pustular forms the prognosis is more grave.

Treatment.—General, rather than local, treatment must be relied upon in the case of dermatitis herpetiformis. The patient's mode of life must be carefully regulated and all worry and anxiety avoided; hence probably the advantages derived from a stay in some quiet country spot or at a spa, either British or Continental. The best of these are probably Harrogate or Strathpeffer, Gastein, Kissingen, or Schintznach. The diet must be ample and nutritious, but non-stimulating. Alcohol is certainly deleterious in most cases and tends to increase itching; the moderate use of tobacco, on the other hand, appears often to be distinctly useful. Milk is extremely valuable as a food, and if well supported should be taken in considerable quantities during the day. Constipation must be carefully guarded against; it is best combated by aperient, bitter, or sulphurous waters taken first thing in the morning, and preferably warmed. The value of arsenic has been variously estimated; Hutchinson reckons it as high, but this is not the opinion of the writer nor of the majority of dermatologists. When the eruption is in its early stages it is certainly aggravated, like many other skin affections, by arsenic, but when on the wane it may perhaps be benefited by the drug cautiously administered. Arguing by analogy it also seems probable that when administered between the attacks arsenic may tend to diminish the tendency to relapses. In similar circumstances small doses of iodide of potassium seem sometimes useful. In some cases quinine in full doses is certainly beneficial. Crocker recommends the use of full doses of belladonna, beginning with fifteen minims of the tincture, and increasing it to thirty minims three times a day. Any evidences of a gouty tendency must be treated by dieting, alkalies, and diuretics; it is in such cases that baths are of special service. To control itching no drugs are more valuable than phenacetin and antipyrin in full doses. There is no valid objection to the use of chloral and bromides to obtain sleep, but morphia is decidedly contra-indicated as tending to increase irritation. A prolonged warm bath at bedtime containing sulphate of potassium (℥ii-℥iv), borax (℥iii), bicarbonate of soda (℥v), bran, linseed, or size (2 to 3 lbs.), in 30 gallons of water, with or without a little liquor. Carbonis detergens often alleviates itching and procures a good night's sleep. Constant rest in bed is in itself beneficial, probably by securing comparative uniformity of temperature.

Locally Duhring warmly recommends sulphur ointment, and his opinion is endorsed by Stephen Mackenzie; it must be vigorously rubbed into the skin, the vesicles and blebs being ruptured. Ichthyol has been warmly advocated in some quarters; internally in five-grain doses, either as capsule or pill, increased up to twenty grains or more three times daily, it is really of service. Its disagreeable odour renders it seldom tolerated in this country as an external application. It may be painted on in aqueous solution from 5 to 25 per cent, or applied as a dusting powder or ointment. The following formulæ are convenient:—

℞ Ichthyol, gr. xx.
Resorcin, gr. x.
Pulveris amyli.
Magnesii carbonatis, āā ℥ss.
Misce. Fiat pulvis.

℞ Ichthyol
Camphoris, āā gr. x.
Olei amygdalæ dulcis, ʒj.
Adipis lanæ, ℥j.
Misce. Fiat unguentum.

In the erythematous form, when few or no vesicles are present, dressings soaked in the following are useful:—

℞ Resorcin, gr. vij.
Glycerini, ♏ xx.
Spiritûs coloniensis, ʒij.
Spiritum vini rectificati, ad ℥j. Misce. Fiat lotio.

The writer has found nothing more useful than weak lead, tar, carbolic or naphthol lotions, but sometimes oily applications containing these drugs are more grateful to the patient.

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Dermatitis Repens.—*Definition.*—A spreading dermatitis, usually following injuries and commencing almost exclusively in the upper extremities.

There is generally a history of an injury to the skin of some part of the hand, though often the injury may be so trivial that its occurrence may have to be carefully inquired for. Vesicles or a bulla develop and rupture, resulting in complete denudation of all the upper layers of the epidermis, the surface being intensely red, and oozing a clear or turbid fluid from numerous points on the surface. The denudation extends peripherally by the epidermis at the border, being undermined and raised up by exudation, forming a sodden ragged collar which can be readily cut away, but nevertheless extension continues, and the disease may travel all up the first affected limb or stop short at any point. Less frequently it extends across the trunk to the other extremity or rarely all over the body. The parts first affected may heal slowly, leaving the skin intensely red and tender. The extension may be very slow—$\frac{1}{8}$ to $\frac{1}{4}$ of an inch a week—and drag on for many months and even for years, or the extension may be at a much more rapid rate. The sensory symptoms are burning and tension rather than itching, and after healing great and persistent tenderness.

A closely allied condition, if not a mere variant of it, are the cases described by Hallopeau, under the title "Acrodermite continue," and by Frèche and Stowers, who acknowledge their resemblance and alliance to dermatitis repens, while Hallopeau considers them separate affections. The actual lesions of the skin on the extremities are practically identical, but Hallopeau attaches importance to the following differences. In Dermatitis repens the disease, if it extends beyond the hands, does so by direct extension of the border, while in Acrodermatitis it is by the formation of fresh foci, and the large areas are formed by their coalescence.

In acrodermatitis there is no permanent healing of the older diseased areas, and the cases go on for years with little or no improvement. The nails are damaged and may be shed and the oral mucous membrane may be affected. An antecedent injury has been present in some but not all of the cases.

Pathogeny.—The theory most consonant with the clinical facts is that there is a peripheral neuritis with secondary microbic invasion of the damaged area, but Hallopeau only admits the microbic origin of acrodermatitis.

Diagnosis.—Eczema is the disease for which dermatitis repens was mistaken until differentiated by the author. The differences are the extension at the periphery by the constant fluid exudation under the border, which forms a well-defined margin. The persistence of the lesions for long periods with slow but constant extension, and the difficulty of healing the denuded surface as well as of checking extension, and the rarity of the formation of fresh foci of disease far away from the original area. Further, for a long time the affection is limited to one extremity.

Treatment.—The disease is very refractory to treatment which need only be local. The most successful plan has been to cut away the undermined epidermis border and paint once a day for ten days with a ten per cent solution of permanganate of potash. The constant application of lint soaked in lactate of lead lotion (Lig. Plumbi Subacet, ʒi. Lactis ℥ij.), has also been successful. Hallopeau paints with a strong solution of nitrate of silver, but the author has not had a good result with this, and Hallopeau has only had amelioration and never a definite cure.

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Dermatitis Traumatica et Venenata.

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CASES of dermatitis from these causes arise from one of three circumstances:—(1) From the pursuance of some employment which exposes the patient to the action of the noxious agent; (2) From the accidental exposure of the patient to some such agent; (3) Feigned eruptions.

Putting aside for one moment those substances which are such powerful irritants or corrosives as to cause a practically immediate destruction of all tissues with which they come into contact, there are some points of interest common to nearly all the irritants of the skin as regards their action in producing a dermatitis. The first peculiar point in connection with occupation or accidental dermatitis is the varying degree of susceptibility found in different people. Thus to poisons which act with the greatest severity upon some persons others may be partially or even completely immune. White relates the case of a child of six years old who died from the effects of severe ivy poisoning produced by having his skin rubbed while wet by the hands of a boy who had been rooting up plants of the poison ivy. This case is rendered still more remarkable by the fact that the boy had previously washed his hands thoroughly, under supervision, first with hot soap and water, and afterwards with vinegar. The boy who had been working with the plants had a full and apparently permanent immunity to the poison.

Secondly, some persons who are more or less susceptible to the irritant at first may be variously affected on prolonged exposure to its action, becoming sometimes immune to its effects, or sometimes more susceptible. In the second case it is, of course, imperative that the injurious occupation be entirely given up for a time, and if after a complete recovery the patient returns to his employment, he may be attacked again either at once or after a long period of immunity. For convenience of description the forms of dermatitis may be divided into:—

(1) Corrosion of the skin with formation of slough, due to very active chemical substances.

(2) Acute inflammations of the skin usually resembling acute eczema, but in some instances restricted to an urticarial œdema.

(3) Chronic forms of simple dermatitis which are quite indistinguishable from chronic eczema.

(4) More or less characteristic eruptions not resembling eczema.

I. The agents producing dermatitis of this variety are: The strong mineral acids and the caustic alkalies, and some metallic salts, such as zinc chloride, etc. The contact of such compounds with the skin is usually the result either of accident or design, since it is obviously impossible that any employment could be followed which entailed constant exposure to the action of such destructive substances. In the case of the acids the effects are usually more circumscribed than in that of the caustic alkalies, owing to the fact that they all cause a coagulation of the tissue albumins and so are more or less self-limited. In all cases the result of the action is to cause a slough surrounded by an area of intense inflammation. After separation of the slough an ulcer of varying extent and size is left, which is usually slow to heal, and has a special tendency to leave an hypertrophied scar. The reaction of the slough is naturally intensely acid where caused by the action of the strong acids, and in the case of nitric and sulphuric has a yellow colour, more pronounced in the case of the former than the latter. The slough left by the action of the caustic alkalies is of a less hard consistency, and is, of course, strongly alkaline in reaction. The colour is usually of a dirty grayish white.

Treatment should be directed towards the free dilution of the poison if seen early enough, and the neutralisation with a substance of the opposite chemical reaction. After separation of the slough the resulting ulcer should be dressed with some bland ointment, such as Lister's boric acid ointment.

II. The agents causing acute dermatitis of the second class may be divided into:—

(*a*) *Animal.*—Jellyfish and allied species, hairy caterpillars and stinging insects, such as hornets, wasps, bees, etc. which all cause eruptions of the urticarial type. The silk in cocoon, which causes an irritable eczema-like eruption on the hands of the winders, and cantharides, which causes bullæ if in strong concentration, or a peculiar pustular eruption with hard papules if constantly repeated in weak strengths.

(*b*) *Vegetable.*—Rhus toxicodendron, venenata, diversiloba, and vernicifera, the first three generally accidental, the last occurring in the process of the manufacture of Japanese lacquer, and occasionally in the handling of the finished article. Primula obconica, cherry laurel, aucuba, arnica, juniperus sabina, staphisagria, capsicum, piper nigra, oil of croton, mustard, turpentine, and thapsia. The above list, which does not pretend to contain the name of every vegetable substance causing dermatitis occasionally, includes those that are most likely to be met with in practice. Besides these there are many members of the family Urticaceæ, as instanced by the common stinging nettle, which produce an urticarial eruption.

(*c*) *Chemical Compounds.* — Certain aniline dyes, as found in clothing, said to be due in every case to arsenic as an impurity, but probably often due to the mordant, which may be arsenic or potassium bichromate. In this connection it may be mentioned that a very severe eruption was found on the arms of some workmen, and traced to the presence of zinc chloride in the clothing. Antimonial salts cause an acute papulo-pustular eruption, but this is not often met with now except in the case of feigned eruptions. Toilet articles and cosmetics have been not infrequently found as causes of an outbreak of eczematoid eruption, especially hair dyes which contain pyrogallic acid or nitrate of silver. Neisser traced several cases of obstinate eczema of the lips to the use of a dentifrice containing many aromatics and some salol. Pyrogallic acid is sometimes also the cause of eruptions on the hands of photographers, but as they are often handling numerous other chemicals, it is difficult to find out exactly the cause in any given instance. Phenyl hydrazin has lately been found by a chemist experimenting with it to give rise to a very acute vesicular eruption resembling eczema. Lastly, many substances used in surgical dressing are apt to cause acute dermatitis, such are iodoform, carbolic acid, and collodion, which last sometimes causes blisters wherever applied.

(*d*) *Physical Agencies.*—X rays (see "X Rays").

The eruptions belonging to this class fall into two subdivisions, namely, the urticarial or œdematous and the true inflammatory. As types of each respectively the lesion produced by the common stinging nettle and that produced by one of the poisonous plants, Rhus toxicodendron, will be described.

A. *The former* needs only the shortest notice, as the rash is almost always quite evanescent in character. It must not be forgotten, however, that in the case of some of the tropical plants the effects are much more lasting and severe, and that with some of the jellyfishes eruptions have occurred which, although beginning as urticaria, ended with gangrene. The symptoms of the urticarial class are then, first, shortly after the contact with the poisonous body, a circumscribed hyperæmia of the irritated spot associated with sharp burning. In a few moments there occurs an exudation of serum into the hyperæmic area, with the result that a pinkish swelling is produced which changes to a yellowish white as the tension in the œdematous papule is gradually raised, and the capillaries are closed by the surrounding pressure. After remaining in this state for some time, usually about half an hour, the exuded serum becomes gradually reabsorbed, and nothing remains but a slight passive hyperæmia to mark the spot where the reaction occurred. If the lesion is situated on some spot where the subcutaneous tissue is very loose, such as the eyelid or scrotum, the swelling is apt to be very much greater and may completely close the eye. *Treatment* consists in the application of evaporating lotions if the pain is very severe.

B. The *acute eczematoid dermatitis* commences as a local hyperæmia or erythema of varying extent. Compared with the erythematous stage of so-called idiopathic eczema this traumatic erythema will be generally found to be more brilliant in colouring and more acute in onset, thus resembling erysipelas. In some cases the disease may go no farther than this, the hyperæmia subsiding after a few hours, and leaving nothing behind but a slight, yellowish discoloration due to diapedesis of red blood-corpuscles, to be followed by an insignificant desquamation of the damaged epithelium. More frequently, however, the hyperæmia is rapidly followed by serous exudation into the corium and, later, into the epidermis itself. This is shown clinically by a marked thickening and swelling of the skin, accompanied by obliteration of the normal folds, and by a slightly translucent appearance in those situations where the skin is thin and the subcutaneous tissue loose, such as the eyelids, penis, and scrotum.

After the exudation of serum has gone on for a certain time the fluid begins to pass upwards into the epidermis, distending the intercellular canals, rupturing the connecting prickles, and pushing aside the cells of the mucous layer so as to form small vesicular cavities. Most of the vesicles thus formed burst, either from pressure of the contained fluid or from external violence. The condition then found is one of intensely inflamed skin covered only by the moist layers of the epidermis and freely pouring out a straw-coloured fluid which dries into gummy crusts on the surface. If the skin be no further exposed to the action of the irritant the exudation of fluid gradually diminishes until it ceases altogether, a new horny layer is formed, and the surface gradually returns to the normal. Frequently the exudation into the mucous layer is so rapid as to raise the horny layer in large areas, and then bullæ are the result, or in other cases where the irritant causes a marked emigration of leucocytes, the vesicles may become quite cloudy and purulent, notably so in the cases of antimonial salts and croton oil. In some cases, again, the inflammation may become so intense as to cause death of the tissues of the papillary layer, in which case ulceration will take place, and the disease can only terminate by the formation of a scar. This, it should be noted, however, is by no means a frequent occurrence in the acute eczematoid forms of dermatitis, but is much more often found in the more chronic cases of irritation by some active chemical agent. As regards the duration of the acute forms of dermatitis no exact time limit can be given. The effects of the poison may be limited to a slight and evanescent redness which passes off within a few hours, or there may be considerable exudation with vesicle formation, in which case recovery will be unlikely to be complete in less than a fortnight, or if the disease reaches a high degree of severity, even without ulceration, it will generally last several weeks.

In addition to this one must remember that some cases of apparently simple acute traumatic dermatitis do not recover of themselves, but after some improvement has taken place tend to pass into a chronic state which is entirely indistinguishable from chronic, idiopathic eczema.

The *subjective symptoms* of acute traumatic dermatitis are in no way characteristic, but are simply those of acute inflammation of the skin, the affection usually beginning with tingling and itching, which is followed by a more or less severe burning and itching as the inflammation progresses to its height.

The *diagnosis* of acute dermatitis from acute eczema is apt to be extremely difficult or impossible. The site and history are important in every case. The sites most usually affected are:—The hands and forearms, the face, especially round the eyes, the neck, the scrotum, and the inner side of the thighs. The reason for the localisation on the hands and face is obviously the greater frequency of exposure of these parts, while probably the scrotum and thighs are often attacked from the extreme delicacy of the skin of these parts, so that any chance contact with hands covered with the irritant is almost sure to produce the eruption. The *prognosis* of the acute forms of dermatitis is almost invariably favourable, though cases have been known where death has ensued, probably from absorption of the poison through the skin, denuded as it is of its protective horny layer.

The *treatment* should be that for any extremely acute inflammation of the skin. Steps should be taken to prevent any further exposure to the influence of the irritant. If the eruption is extensive and severe the patient should be kept in the recumbent posture, so that the circulation may be rendered as quiet as possible, and only a light diet should be allowed. No internal treatment has any direct action on the course of the eruption, but it may be of advantage to give a dose of calomel at the commencement of the attack, as this will at least tend to expedite the excretion of any of the poison which may have been absorbed.

Locally, lotions are of most service in the early stages, such as diluted black-wash, lead, or calamine lotion. If itching is severe a very small percentage of carbolic acid may be added to the lotion. In the United States and Canada, where the disease is of much more common occurrence than in this country, owing to the greater prevalence of poisonous plants, the fluid extract of Grindelia Robusta, diluted one in thirty, is in great repute. After the cessation of the discharge and the more active symptoms of inflammation, recourse may be generally had with advantage to bland pastes or ointments, but in no case must any stimulating application be used unless the eruption shows signs of becoming chronic. One of the best soothing and protective applications will be found to be a cream made up of equal parts of zinc oxide, almond oil, and lime water, with fifteen grains of anhydrous lanoline to each ounce of the mixture. This application is cleanly, and is also cooling from the evaporation of the water, while it contains just sufficient grease to soften the inflamed skin and render it supple, thus obviating the uncomfortable feeling of stiffness and the liability to fissure which are apt to be present owing to the insufficiency of the horny layer.

III. CHRONIC DERMATITIS.—This is caused by the prolonged action of substances, either of less virulently irritating properties, or in a greater state of dilution than those which cause the acute forms of dermatitis. The offending body may act simply *mechanically*, as in the case of fine powders, accounting for the rashes found in potters due to the fine clay, in millstone and quarry cutters from particles of stone, in glass-paper makers, pearl cutters, knife and needle grinders, etc. Or the substance may be irritating both *mechanically and chemically*, as the finely-powdered sugar which irritates the arms of grocers, or, lastly, the irritant may act chemically only. In this last class the offending agents are almost innumerable. In contradistinction from the acute form of dermatitis the chronic form is almost confined to people whose pursuits bring them into daily contact with the offending substance. The commonest cause of all is the constant immersion of the skin in water containing an alkali, such as is present in most soaps. Another very frequent cause is the washing up of utensils which contain decomposing alcohol and weak acids, such as are found in the dregs of beer and wine glasses, hence the disease in barmen and waiters; also in cheap methylated spirit, such as is used in dissolving varnishes. Repeated contact with most metallic salts in the moist state will in time irritate the skin so as to produce a chronic dermatitis, and hence arises the disease known as galvaniser's eczema, due generally to the ammonio-nickelic oxide in the bath. Almost all the aromatic oils and perfumes are capable of inducing a chronic dermatitis when constantly used, and are generally found in face washes, powders, etc. Also many drugs are responsible for cases of skin disease in those who prepare them, instances being quinine, aconite, podophyllin, rue, vanilla, and oil of bitter orange.

Chronic dermatitis from any of these causes is usually of the dry lichenoid type. The skin is diffusely reddened and infiltrated, the epidermis is thickened generally, is of a harsh, dry character, and contains much less grease than normally. Consequently the normal folds of the skin are much deepened, and are apt to form troublesome fissures from the want of elasticity and proper cohesion of the diseased horny layer. The mouths of the follicles are often slightly gaping and hyperkeratotic, and the lanugo hairs are stunted and broken. Vesicles are found here and there from time to time, and are usually situated rather deep down in the epidermis. Secondary pyogenic infection of the fissures is very common.

The eruption is apt to spread beyond the points of actual contact with the irritant, and this fact has given rise to much discussion. Some observers hold that this spreading beyond the actual points of contact is proof that chemical irritants can call forth a true eczema, while others contend that the spreading is caused by the inoculation of the eczema virus upon the already damaged skin. Be this as it may, it is found that chronic dermatitis from external irritants is almost invariably symmetrical, that it often lasts long after the cause has been removed, and shows little or no tendency towards spontaneous recovery, and that it is especially liable to attack the sites of predilection of idiopathic eczema.

The *treatment* of this form of eruption is of course primarily to remove the source of irritation. After this has been done it will be generally found necessary to apply some bland emollient preparation for some time until the more actively inflammatory symptoms have subsided and any fissures present have healed. One may then proceed cautiously with some weak form of stimulant of the class known as reducing agents, and perhaps one of the best will be found to be Pick's salicylic acid soap plaster, which should be spread rather thickly upon old, coarse linen, and kept continuously applied day and night. The general principles of treatment of chronic eczema apply equally well to this artificial dermatitis, and need not be gone into in detail.

IV. The fourth class of eruptions, those that are more or less characteristic, contains only a few members.

The first group consists of those caused by arsenic, antimony, potassium bichromate, and potassium cyanide. The first three of these drugs may all of them produce an acute eczematoid dermatitis, though Richardson stated that potassium bichromate had no action on the sound skin. The feature common to them all, however, is that they are liable to produce very obstinate ulceration if they come into contact with even the slightest abrasion of the epidermis. The first three substances are all occasionally used, either in the preparation or the mordanting of aniline dyes, and potassium bichromate is also present in certain kinds of wood polishes. Potassium cyanide is used by photographers, but on account of its known poisonous action its effects upon the skin are less often seen owing to the care with which it is handled.

As regards the diagnosis of these eruptions arsenic should be suspected if there is either digestive disturbance or inflammation round the eyes, while bichromate stains the nails and skin a characteristic yellow colour. There are no special characteristics which would enable one to diagnose the presence of the other two poisons.

A very characteristic eruption is seen on the skins of tar workers, and probably the same is caused by allied substances, such as paraffins and soot, owing, in the case of the last-mentioned substance, to the traces of coal tar contained in it. The eruption appears on places where the skin is brought into contact with the offending substance, in the case of the tar worker, hot anthracene oil, or more rarely creasote oil. It is greatly dependent on the habits of personal cleanliness of the individual, those who take care to thoroughly wash off all traces of the irritant on leaving work suffering very much less than those who are careless in this respect. The sites of predilection are the face, especially the hairy parts, the backs of the hands and arms, though occasionally sparse lesions are found on the palms, and the scrotum.

The earliest lesion appears to be the plugging of the follicles with inspissated tar products. The irritating action of this obstruction, probably partly mechanical and partly chemical, causes a rapid overgrowth of the cells around the follicle, so that the mouth now contains a blackened horny plug. Below this suppuration may occur, or the plug may be detached by simple mechanical movements. In either case a small depressed scar is the result, so that the arms have a honeycombed appearance, especially when viewed in an oblique light. Besides these scars, however, there are always present numerous little horny plugs which have not become expressed. The hair is usually maintained since the destruction is too superficial to affect the bulb. Associated with this stage of the eruption is also a series of red and very slightly thickened spots, which are seen under a lens to consist of dilated blood-vessels, and are, in all probability, due to the repeated hyperæmia from the splashes of the hot oils. At all events these hyperamic spots are not acutely inflammatory since they last for years. In some cases the follicular plugs, instead of being removed by suppuration or otherwise, grow to a considerable size and then agglomerate to form the so-called tar mollusca. It is plain that there must be considerable proliferation from the first of the cells at the mouth of the follicle, in order to produce that hyperkeratosis which is one of the earliest appearances. Later this proliferation apparently affects the deeper cells, and then these grow out sidewise beneath the surrounding epidermis so as to produce a hard base and a pearly edge, thus completely simulating rodent ulcer. The growth is, however, not yet malignant, though at any moment it may become so, and in most cases if left alone will slough out, leaving an ulcer which heals and produces a cribriform scar not unlike that left after vaccination. At the same time, in addition to the molluscum form of tumour, there is another, the common flat wart, the evolution of which is not quite so clear. On examining the affected skin in early stages, however, it can be seen that there is general hyperkeratosis of the parts between the follicles, though not to so marked an extent as of the follicles themselves, and it is probably from this interfollicular skin that the common wart is developed. These warts may also become the seat of malignant disease, taking on the characters of true epithelioma. It may be remarked that, in the case of sweep's cancer of the scrotum recently seen by the writer, a careful search revealed the presence of very numerous small, flat warts on the ulnar borders of the flexor surfaces of both forearms.

The *treatment* of this affection should be in the first place of a preventive kind. The men in tar works should be protected as far as possible from the splashes of the hot liquids, and should be in all cases encouraged to observe scrupulous cleanliness after cessation of work. As regards curative treatment, when the eruption has once developed there is little to be done at first. The workmen all know themselves that if they pick off the little horny projections they are liable to aggravate the disease. If any of the tumours grow to an inconvenient size it is advisable to remove them without waiting for exfoliation, and in any case a sharp lookout should be kept on all growths, so that, should any of them develop malignant tendencies, they may be removed at once.

As has been already noted, a rash strongly resembling that just described has been observed in workers with paraffin, though this agent does not appear to produce the epithelial tumours.

There is, however, another form of eruption occasionally seen on the legs of those men who habitually carry about vessels of petroleum. Usually the right leg only is affected, owing to the fact that the can is carried in the right hand. The right trouser leg gets saturated with the oil, and its action, aided perhaps by the constant friction of the skin, produces a curious bullous eruption situated on a brawny and inflamed base. There is usually a considerable amount of pyogenic infection of the eruption, which soon dies away under soothing and protective pastes.

Recently, a chemical much used as a developer of photographic plates, *metol*, has been found to cause a somewhat characteristic eruption on the hands. The salient features of the eruption are:—A strikingly polished appearance of the epidermis as if varnished; a diffuse, even, cyanotic hyperæmia of the affected parts, almost blue in colour; thickening of the skin itself, with the subjective symptoms of numbness, stiffness, itching, and pain. The eruption somewhat resembles both perniosis and erythromelalgia. From the former it is distinguished by its occurrence in hot weather and its affecting all the fingers evenly, while from the latter it may be easily differentiated by the absence of the characteristic paroxysms of pain. Recovery takes place in two or three weeks after removal of the cause.

Feigned Eruptions.—Lastly, a short description must be given of the feigned eruptions. These have been separated from the main body of artificial eruptions—first, on account of the fact that they are not all true inflammations; and, secondly, because they present some peculiar features of interest. There are two classes of case to be considered, that in which the eruption is produced for obvious reasons, such as to avoid some distasteful occupation, and that in which there is a morbid state of the mind. Patients belonging to the second class should always be most carefully watched for symptoms of mania developing. Thus in one of Shepherd's cases a girl ran out of the hospital ward, where there was no fire, with her clothes on fire.

The types of eruption may be divided into anomalies of secretion, such as red and black sweating, blue concretions at the mouths of the sebaceous follicles, etc., and inflammatory eruptions.

In the former class the number of colouring agents which may be used is almost unlimited, though curiously enough soot seems to be one of the most favourite applications. The pigment is generally made up with grease to make it adhere properly, so that a few drops of benzine on a tuft of cotton wool will clean it off, leaving a perfectly normal skin beneath. The differential *diagnosis* of these eruptions from those of true-coloured secretion is very difficult, and is only to be accomplished either by careful watching, or in those instances in which the substance used can be identified by chemical or microscopical examination, as in a case in which extract of liquorice was used to simulate bleeding points.

In the *inflammatory* class of cases the eruption may be simply a hyperæmia, or it may be more often bullous, vesicular, and pustular, with gangrene and ulceration, or again it may be some deep-seated chronic inflammation of nodular character. Often there is, as in other kinds of hysterical manifestation, some true lesion of the skin or some eruption produced for therapeutic purposes as the forerunner of the feigned manifestation. Thus some patients who have been treated by blistering by one doctor, have afterwards procured preparations of cantharides with which to deceive people. Some of the substances used have been nitric acid, carbolic acid, cantharides, croton oil, tartar emetic, and mustard. The circumstances which should always give rise to suspicion are: incorrigible idleness in men, hysteria in women and girls, the age of puberty, the correspondence of the eruption with no known form of idiopathic eruption, combined with a similarity to the effects of known irritants, and the occurrence on the left side and on situations which are easily reached by the patient. In many cases the irritant will have run on the skin, producing a streak below the patch of inflammation, and the patch itself has often irregular outlines. In doubtful cases the litmus paper should always be used, as a very strong acid reaction of the tissues will remain for days when any of the mineral acids has been used. Sometimes the patch may smell of the corrosive, as in one case where the slough was produced with crude carbolic acid used for disinfecting purposes. Lastly, the area, if showing the chronic inflammatory form, may be carefully wiped with a pledget of cotton wool soaked in soft soap and water, and the material thus obtained tested for arsenic and antimony. Croton oil, one of the favourite applications for producing feigned eruptions, is not easily identified chemically, and its use can only be suspected by the presence of its pustular eruption on the skin and by careful search of the patient's belongings. Other methods of producing curious eruptions are by friction of the moistened skin, sometimes after previous prolonged sucking of the part, thus producing an excoriated wheal, or by the application of heat for so short a time that no true bulla formation is produced but a superficial destruction of the epidermis with subjacent hyperæmia. Many cases of spontaneous gangrene of young women have been published on the Continent, but on reading the reports of these cases the writer is convinced that most, if not all of them, were really instances of feigned eruption.

The *treatment* in all doubtful cases should be the careful dressing of the affected part in such a manner that the patient shall be unable to get at the place. In such cases the eruption has often been found to occur immediately beyond the dressing, a fact which, if repeated, is practically diagnostic. Exposure of the patient, when detected, is not always satisfactory in its results, since it is usually impossible to convince the relations, and the patient only gets more sympathy. Sometimes the patient herself can be quietly lectured with better results, but it is probably always better to put her under a rigid course of treatment directed against the mental unsoundness.

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Dermoid Cysts. *See* TUMOURS.

Diabetes Insipidus.

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DIABETES INSIPIDUS is a disease characterised by a prolonged morbid increase in the secretion of a urine free from sugar.

ETIOLOGY.—Diabetes insipidus is a comparatively rare condition. During a period of six years twenty cases were under treatment in the wards of the Royal Infirmary, Edinburgh. According to the statistics of Roberts, Strauss, and van der Heijden, the disease is most common during adolescence, early manhood, and middle life. Sex seems to play a not unimportant part as a predisposing factor, males being twice as frequently affected as females. A marked family predisposition may exist, the disease running through neuropathic families or alternating with other nervous or mental troubles. Thus Griesinger in one instance found that seven blood relations of the patient were epileptic, in two instances the patient himself was epileptic, in one instance both the patient and his friends were epileptic. Gee notes a family where in four generations of 23 patients, 11 suffered from diabetes insipidus; Orsi, a family of 9, 6 of whom were affected; and Weil a family of 21, 20 of whom developed the complaint. The causal relationship between syphilis and diabetes insipidus is not yet quite clear. In most cases the condition must be ascribed to cerebral gumma or endarteritis, but a number remain where the exact condition cannot be substantiated. In brain diseases tuberculosis must not be forgotten in diabetes insipidus in childhood, and two cases have been described where chronic hydrocephalus existed.

The acute infectious diseases, gout, trauma of the head or abdomen, mental shock and worry, exposure to cold, a bout of drunkenness, neglect and improper feeding, with consequent malnutrition in children, may all have a causal relationship to the disease.

Symptoms.—Two prominent and characteristic symptoms are present in the disease—an increase in the amount of urine excreted and a marked and distressing thirst. The onset of the disease may be insidious or sudden, there is complaint of increased frequency of micturition and increase in the amount of urine passed, distressing during the day, and preventing proper sleep during the night. The amount of urine is enormously increased, as much as fifteen to twenty pints being passed in twenty-four hours. Except in the later stages of the disease the amount of urine excreted exceeds considerably the amount of fluid ingested, the surplus being made up from the food and the tissues of the body. When, however, a certain stage of the disease is reached the tissues become dehydrated, and this disproportion is lost. The urine is pale in colour with a yellowish or greenish tinge. The reaction is faintly acid or neutral. The specific gravity is always low—1002° to 1005°. Glucose is absent, but inosite or muscle sugar is frequently present, but its presence is not characteristic, as it may be found in chronic interstitial nephritis, and even in health after an excessive amount of water has been drunk. As a rule albumin is absent, except in the later stages of the disease, when it may be present as the result of the general dyscrasia and disorganisation of the kidneys. The urine on standing may become turbid from the deposit of epithelial cells; crystals of oxalate of lime may be present, and phosphates may form from ammoniacal decomposition. The percentage of total solids is small, but the total daily excretion of nitrogen may be slightly increased. The chlorides and phosphates are increased. The thirst is excessive and distressing. Enormous amounts of fluid can be swallowed by the sufferer. Trousseau recounts the case of a young man who daily drank forty litres of fluid and passed forty-three litres of urine. Under treatment, when unable to get water the thirst became so intolerable that he drank the contents of the chamber-pot.

The mouth, tongue, and fauces are dry and parched. The appetite may be unaffected, but it is usually increased, and in the early stages of the disease may be voracious. Digestion in the earlier stages of the disease is normally performed. The bowels may be constipated. The temperature is as a rule subnormal. The skin is dry and harsh. The nervous system is disturbed. When the condition is due to a gross cerebral lesion paralysis of cranial nerves with hemianopsia may be present. The sensory functions are usually abnormal, thus pains in the back and legs and a tendency to headache, tingling and itching of the skin may be present. Amblyopia is common without any discoverable lesion. The mental functions are below par, the patient being irritable and depressed. Insomnia is distressing. The sexual powers are enfeebled.

The progress of the disease is variable. At times the condition is so slight as to be regarded more as an annoyance than as a disease. In more severe cases progressive deterioration in nutrition sets in, the muscles waste, the appetite diminishes, digestion fails, and attacks of diarrhœa supervene. The patient becomes exhausted, drowsy, and gradually comatose, unless carried off by an intercurrent attack of hypostatic pneumonia.

Diagnosis is not as a rule difficult. The increased excretion of water in diabetes mellitus, intermittent hydronephrosis, chronic interstitial nephritis, and in hysteria, may occasionally simulate that symptom in diabetes insipidus, but these conditions are otherwise readily differentiated.

The *prognosis* in diabetes insipidus is very variable. Each one must be judged upon its own merits. At times the health does not suffer appreciably. Cure may sometimes follow an intercurrent affection such as measles. Idiopathic cases may last for many years, and terminate in a gradual loss of nutrition or in an intercurrent attack of pneumonia or with a slowly progressing tuberculosis. Once anorexia sets in the end is not far distant. When a gross cerebral lesion is present the prognosis is bad. When the disease appears in childhood the general development may be retarded and deficient.

The pathology of diabetes insipidus consists essentially of an error in the controlling power of the vasomotor nerves of the kidney. In animals polyuria has been shown to follow stimulation of the floor of the fourth ventricle or cerebellum. In man the stimulation may result from peripheral irritation; an abscess of the external auditory meatus has been known to produce marked polyuria, which subsided on evacuation, to return when the free discharge of pus was obstructed, to disappear again as healing took place. In animals section of the great splanchnic nerve is followed by persistent hypersecretion from the kidney on the same side. In man Schapiro has described degeneration and atrophy of the ganglionic cells of the semi-lunar ganglia, and degeneration and destruction of the axis cylinders of the great splanchnic nerve in diabetes insipidus. Various lesions of the brain may be present, as tumours, tubercle, degeneration and softening following arterial changes. There is no characteristic lesion in the kidneys, though in long-standing cases secondary changes may take place as the result of the prolonged excessive secretion of urine.

Treatment.—The treatment of diabetes insipidus, founded as it is upon an imperfect pathology, is not entirely satisfactory. When a definite dyscrasia exists, such as tuberculosis or syphilis, as a causal factor, treatment must be directed to its amelioration by suitable remedies. Apart from this, treatment by drugs has, for the most part, been directed to influencing the condition through the nervous system. Valerian was advocated by Trousseau in enormous doses of 10 grms. per diem, progressively increasing to 30 grms. Given thus it inevitably results in gastric disturbance. Smaller doses of ʒss. to ʒj. of the tincture thrice daily, with camphor water or spirits of chloroform to disguise the taste, produce a decided, but unfortunately but passing improvement, and any effort at increased dosage is resented by the stomach. Ergot has a decided ameliorating influence, and may be suitably prescribed as ergotin in 2-grain doses in pill with extract of belladonna, given every six hours, the effects being watched lest ergotism be produced. Nitroglycerine, again, has produced decided benefit in a number of cases. Antipyrin may prove useful at night as a sedative, and temporarily diminishing the excretion of urine, and thus permitting sleep. Salts of iron, arsenic, zinc, strychnia, belladonna, nitrate of silver, carbolic acid, and the bromides have all been prescribed with varying success. Drugs, in fact, may diminish the excessive secretion of urine for a time, and have a beneficial effect on the general health; they do not cure the disease. Ergot, bellaponna, and nitroglycerine are most to be recommended. Electricity has had a marvellous curative effect in some instances. It may be applied to the medulla, the positive pole of the galvanic current being applied to the back of the neck by a large electrode, the negative pole, suitably insulated to within a quarter of an inch of its extremity, being passed along the floor of the nostril to reach the spine. The strength of the current should be gradually increased from one to five milleampères, the duration of the application from one to five minutes. The negative pole may be applied to the epigastric region instead of to the posterior wall of the nasopharynx.

Diet and general hygienic measures are of great moment in the treatment of the disease. It is unnecessary and cruel to needlessly restrict the amount of fluid drunk, and only leads to deterioration in the general health. The daily consumption of fluid should be ascertained, and a gradual diminution advised till it is found that the urinary excretion is no longer influenced. Thirst should be alleviated with ice, acidulated drinks, and the use of pilocarpine in small doses. Alcohol, aerated waters, potash water, tea, and coffee must be avoided on account of their diuretic action. Food should be nutritious and easily digested. There is no objection to carbohydrates, nor to proteid food within the limits of the patient's powers of digestion. If restriction be placed upon the amount of food a careful watch must be kept upon the patient's weight. It is better that food should be taken frequently in smaller quantity than in large quantities at longer intervals. Little salt must be taken in the food. In the later stages, when anorexia supervenes, the fluids drunk should be nutritious, as milk, cocoa, whey, light broth.

General hygiene is of importance—gentle exercise, a bracing climate, and, as the temperature is subnormal, warm clothing are all important. In the later stages carefully regulated massage is of great use. Great care should be taken of the skin, which becomes atrophic and unhealthy. Dryness may be relieved by steam or warm water baths. Gentle friction aids nutrition. Intercurrent affections must be treated on general principles. Constipation is frequently troublesome, drastic and irritant purgatives must be avoided from their liability to set up severe diarrhœa. Collection of fæces occurs in the lower bowel, and is best treated by large enemata, massage to the abdomen, aloin. $\frac{1}{10}$ grain in pill after meals, or fluid extract of cascara sagrada in half-drachm doses at night.

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Diabetes Mellitus.

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Diabetes Mellitus is a disease in which grape sugar is persistently excreted in the urine. The term cannot be applied, however, to all cases in which sugar is detected in the urine. The cases of temporary glycosuria are to be excluded; so also are cases in which the sugar excreted is not grape sugar, as for example, lactosuria and pentosuria. The name diabetes mellitus is applied by many writers to all forms of *permanent* glycosuria. Others use the term chronic glycosuria for the milder forms of the disease, in which grape sugar is present in the urine but other symptoms slight or absent, and reserve the term diabetes mellitus for the more severe forms.

Physiological Considerations.—The carbohydrates derived from the products of digestion are conveyed by the portal vein to the liver. This organ contains a carbohydrate, glycogen, which is greatest in quantity when a carbohydrate diet is given. What the exact function of the liver may be, whether it is constantly paying out a small quantity of sugar into the general circulation, or whether the function of the liver and the intestinal villi is to prevent sugar passing into the general circulation, is a disputed point which will be discussed in the article on the liver functions.

Pavy thinks that if sugar were continually being passed into the general circulation, the urine would always contain sugar in quantity. He believes that any excess of sugar in the blood is always eliminated in the urine.

It has been shown, however, that the sugar in the blood disappears when the liver is excised (Minkowski), or when the vessels of the liver are ligatured (Bock and Hoffmann, Seegen).

Intravenous injection of large quantities of sugar have not been followed by glycosuria (Biedl and Kraus). Also subcutaneous injection of certain kinds of sugar—dextrose, lævulose, and galactose—were not followed by glycosuria; but after the subcutaneous injection of cane and milk sugar, the whole of these substances was found again in the urine (Voit).

Experimental Diabetes and Glycosuria.—In this article it is only possible to very briefly mention some of the more important results of experiments on animals.

C. Bernard produced diabetes by puncturing the floor of the fourth ventricle. It appears probable that changes are thereby produced in the hepatic cells, and that the impulses which cause them pass along the splanchnic nerves. By other experimental lesions of the nervous system diabetes may be produced, as for example, by division of the medulla (Pavy), injury of the vermiform process of the cerebellum (Eckhard), injury of various parts of the pons and posterior columns of the spinal cord (Schiff), centrifugal vagus irritation (Arthaud and Butte). Pavy produced diabetes by injection of defibrinated arterial (oxygenated) blood into the portal vein. Diabetes can also be produced both in man and animals by the administration of phloridzin (V. Mering).

Alimentary, puerperal, and symptomatic glycosuria, as well as glycosuria produced by phloridzin and chemical substances, will be considered in the article on glycosuria.

Pancreatic Diabetes.—It was not until 1889 that it was found by Minkowski and v. Mering in Germany, and by De Dominicis in Italy, that total extirpation of the pancreas in dogs is followed by diabetes. Partial extirpation, *i.e.* when one-quarter or one-fifth of the gland is left behind, does not produce diabetes. Even when the pancreatic duct is ligatured and the remaining piece of the pancreas has no connection with the duodenum, still diabetes does not follow. Minkowski has shown the relation of the pancreas to diabetes by the most striking experiment of transplanting a piece of the gland and grafting it under the skin of the abdominal wall (external to the abdominal cavity). If the transplanted portion of pancreas (or graft) does not necrose, then diabetes will not occur when the whole of the remaining intra-abdominal part of the gland is removed. But if the transplanted portion of pancreas be subsequently removed, then diabetes occurs.

It is probable that something is formed in the pancreas which passes into the circulation and brings about sugar destruction, or prevents the accumulation of sugar in the blood. Lépine and others believe that this "something" is an internal secretion of the pancreas, which is absorbed by the pancreatic lymphatics and veins.

Etiology and Etiological Relations.—The disease is more common in males than females; the liability of the two sexes is about equal in the early period of life, but after the age of 30, males are more frequently affected than females. The disease is more common in adults. In private practice it is most frequently met with between the ages of 50 and 60, but in hospital practice there is a larger proportion of young diabetic patients.

The following table gives the age and sex of 100 cases of diabetes in Manchester (mostly hospital patients):—

	AGE IN YEARS.						
	10-20	20-30	30-40	40-50	50-60	60-70	Total.
Males	6	12	14	18	9	3	62
Females . . .	6	13	8	3	3	5	38
	12	25	22	21	12	8	100

The percentage at various ages recorded by several authors is as follows:—

	Under 10.	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Grube . .	...	1·7	2·8	11·2	23·1	39·5	18·1	3·4
Seegen . .	0·5	3	16	16	24	30	10	0·5
Frerichs . .	1	7	10	18	25	26	11	1

The disease is comparatively rare. At the Manchester Royal Infirmary during the twenty years 1875-95, the number of medical in-patients was 27,721, and of these only 272 suffered from diabetes, *i.e.* only 0·9 per cent.

In India, Ceylon, South Italy, and Malta, the disease is much more common than in most other countries. In India it is more common among the Hindus than the Mohammedans. It is said to be more common amongst the Jews, but probably it is the wealthy Jews who chiefly suffer.

The reports of the Registrar-General show that in England the mortality from the disease is steadily increasing. A similar increase in the mortality has been noted in France, Denmark, and the United States.

PREDISPOSING AND EXCITING CAUSES.—Sometimes there is a *family history* of diabetes (13 per cent). Brothers or sisters occasionally suffer: sometimes an uncle or aunt of the diabetic patient has been similarly affected; but it is very rare to find that the father or mother have had the disease.

After *external injuries* temporary glycosuria sometimes occurs, and occasionally a true diabetes follows. It is evident that there must be some other factor in the causation. Still sufficient cases are now on record to show that probably an injury is sometimes the exciting cause (6 per cent of diabetic patients). In at least half of the cases of traumatic diabetes, the head has been the seat of the injury.

Numerous striking instances are on record in which diabetes has rapidly followed *fright, violent passion*, or *mental emotion:* also in many cases the symptoms have followed prolonged mental anxiety and worry, owing to loss of money and loss of employment, etc. (this history obtained in 10 per cent of cases). Mental anxiety and over-work associated with the nursing of a sick relative, etc., is sometimes an exciting cause (8 per cent of cases).

Obesity is sometimes associated with a mild form of diabetes in individuals between the ages of 40 and 60. In young persons occasionally great obesity is followed by a severe form of the disease. (Great obesity preceded diabetes in 4 per cent of writer's cases.)

Gout may be associated with diabetes which is usually of a mild form. Such cases are met with chiefly in private practice and are rare amongst hospital patients.

A marked history of *alcoholism* (chiefly beer-drinking) is sometimes obtained (17 per cent of cases), and probably this is an occasional cause of the disease.

An attack of influenza, an acute febrile affection, pneumonia, bronchitis, pleurisy, exposure to wet and cold, the drinking of cold fluid when the body has been very hot, and injury from a lightning stroke, have been regarded as occasional exciting causes of the disease. (Diabetes developed directly after an attack of influenza in 8 per cent of cases collected by writer.) It is possible that in a few cases *syphilis* may be an indirect cause of diabetes by producing cerebral or pancreatic lesions.

Occasionally diabetes follows *pregnancy* or the development of an abscess of the breast (7 per cent of writer's cases). Occasionally diabetes develops during pregnancy, but ceases with the termination of the latter, to recur at a later date. It has been thought by some writers that the climacteric period favours the occurrence of diabetes in women. In at least 15 per cent of the cases *no history of any exciting cause* can be obtained.

Relation between Diabetes Mellitus and Diseases of the Liver.—From the results of physiological experiments, one would expect that there would be some clear relationship between diabetes and pathological changes in the liver; but no definite or constant pathological change is met with in the liver in diabetes, though this organ is sometimes diseased. In 20 cases in Manchester, the liver, as regards size, was enlarged in 11, diminished in 4, and normal in 5. In one of the cases multiple abscesses were present, in another there was cirrhosis, in another fatty infiltration and cirrhosis, in another fatty infiltration and congestion. In the other cases beyond variations in size, the only change was congestion, which was often present. These are the most common abnormalities; they are not constant, and are very often met with unassociated with diabetes.

The glycogen in the liver cells removed by a fine trocar during life was in one case considerable, but in another case it was absent (Ehrlich). At present *pathological anatomy* does not furnish any evidence that diabetes is related to hepatic changes.

Relation between Diabetes Mellitus and Affections of the Nervous System.—It has been already mentioned that diabetes has often followed great mental anxiety, worry, or sudden fright, and there are many points in favour of the connection of diabetes with some change in the nervous system. But important pathological lesions have been met with in only a small proportion of cases. The changes usually recorded are slight, and such as are frequently met with when no symptoms of diabetes have been present during life.

A number of cases are on record, however, in which cerebral changes have been found post-mortem, that have probably been the cause of the diabetic symptoms. In 30 of such cases recorded in literature, there was a tumour in the floor of the fourth ventricle in 4, other changes at this region (such as softening, fatty degeneration, sclerosis, hæmorrhage, cysticercus) in 14, tumour of the medulla in 3, lesion at the base of the brain (tumour, softening) in 3, tumour of the pituitary body 1, cysticercus in cerebellum 1, softening in cerebellum 1, cerebral tumour (tempero-sphenoidal lobe) 1, tumour compressing right vagus nerve 2.

In 14 consecutive cases recently examined by the writer, the medulla and other parts of the brain appeared normal in 9, in 5 naked-eye changes were found. In the later cases the changes were the following: cyst of the cerebellum close to the right vagus root in 1, cerebro-spinal meningitis (probably secondary to hepatic abscess) in 1, minute hæmorrhagic patch in left vagus nucleus in 1, tumour of the pituitary body in 2 (in these two cases symptoms of acromegaly in addition to those of diabetes were present during life).

Careful microscopical examination of the medulla has been made frequently, but usually with negative results. The minute excavations around the cerebral arteries described by Dickinson are now generally believed to be due to the effects of hardening. Frerichs described a marked dilatation of the small vessels of the medulla, and this he regarded as the most important and constant change in the nervous system in diabetes. Of 10 cases of diabetes examined microscopically by the writer, the medulla appeared normal in 4; in 1 there was purulent meningitis; in 5 cases the blood-vessels of the vagus nuclei were much dilated. In 3 of the latter 5 cases there were small hæmorrhages present in the vagus nuclei, twice unilateral, once bilateral. In one of the cases the hæmorrhagic patch could be seen with the naked eye.

Though the examination of the medulla often yields negative results, it is still possible that minute or functional changes may be present in the nerve cells of the vagus nuclei, or at other part of the medulla, which cannot be recognised at present by microscopical examination.

The spinal cord is usually normal, or presents changes which are to be regarded as secondary; but in a very few cases gross lesions have been found which may have been the primary cause of the symptoms (tumour or softening of the cervical region).

The sympathetic nerves and ganglia (cervical and abdominal) have been carefully examined by Hale White and others; often they have been normal, and when changes have been found they have not differed from those which have been frequently met with when there have been no diabetic symptoms during life.

Occasionally a mild glycosuria occurs in association with well-marked disease of the nervous system such as locomotor ataxia, disseminated sclerosis, Graves' disease, etc. But the association of a well-marked diabetes with these affections is very rare.

DIABETES AND ACROMEGALY.—During the last 5 years a number of cases of acromegaly have been recorded in which glycosuria was present, and in some of the cases there has been a well-marked diabetes (2 cases of diabetes associated with acromegaly have come under the writer's observation). Out of 21 cases of acromegaly recorded in literature, 4 were associated with true diabetes, 2 with glycosuria, and in 15 cases the urine was free from sugar. In acromegaly a tumour of the pituitary body is usually present, and cases of tumours of the pituitary body are on record in which diabetes has been present, but symptoms of acromegaly absent.

THE RELATION OF DIABETES TO LESIONS OF THE PANCREAS.—Changes in the pancreas have been described from time to time, ever since Cawley recorded a case of diabetes (in 1788) in which the pancreas was atrophied and contained calculi. Many years ago, Lancereaux drew attention to the pancreatic lesions.

In 24 consecutive cases of diabetes examined pathologically by the writer, the condition of the pancreas was as follows:—

	Cases.
(*a*) Extensive changes (very marked cirrhosis 2, cancer 1, extensive atrophy 1)	4
(*b*) Well-marked changes (cirrhosis 2, lipomatosis 1, atrophy with fatty degeneration and infiltration 1)	4
(*c*) Slight changes (atrophy with slight fatty degeneration 1, atrophy out of proportion to the general wasting 2)	3
(*d*) Atrophy, but only in proportion to the general wasting	5
(*e*) Pancreas normal, macroscopically and microscopically	8
	24

In the first 4 cases of the above table the pancreatic changes were so extensive that, bearing in mind the result of total extirpation of the gland in animals, there can be little doubt that the diabetes was due to the pancreatic lesion.

In the second group of cases it also appears probable that diabetes was due to the pancreatic changes; in the third group the relation is uncertain; and in the last two groups of cases either the diabetes was not dependent on the pancreatic disease, or the pancreatic affection, if present, was a functional one which could not be recognised macroscopically or microscopically.

Many cases of diabetes associated with pancreatic disease are now on record, and various extensive changes have been found, such as cirrhosis and fibroid changes, fatty infiltration and degeneration, calculi, cancer, cysts, and marked atrophy. Often the pancreatic tissue has been almost absent owing to the changes mentioned.

It is very improbable that all these varied pancreatic changes should be the result of diabetes; and when we consider the remarkable results of experimental removal of the pancreas, it appears very probable that, in certain cases, diabetes is directly due to pancreatic disease.

There are two objections to the pancreatic theory of diabetes in man. The first is that *glycosuria is absent* in *many cases of diseases of the pancreas.* But it is important to remember that in extirpation of the pancreas in animals, if a small portion of the gland should be left behind, no diabetes follows. And in man, in disease of the pancreas, often only a portion of the gland is affected.

The second great object to the pancreatic origin of diabetes is the fact that the *pancreas is not affected in all cases of diabetes.* In one-third of the cases the pancreas is normal macroscopically and microscopically. Hence in these cases either the lesion causing the diabetes is not in the pancreas, or the pancreatic affection is a functional one.

It is conceivable that arterio-sclerosis may cause diabetes by producing changes in the pancreas or in the nervous system; and several instructive cases of diabetes are on record in which marked pancreatic changes were found post-mortem that were apparently the result of arterio-sclerosis.

DIABETES OF ENDOGENOUS ORIGIN.—In many cases of diabetes the most careful inquiry fails to reveal any exciting cause, and often the most careful examination of the brain, pancreas, liver, and other organs fails to reveal any changes except what are secondary or accidental.

It is quite possible (as suggested by Strümpell), that some cases are entirely or almost entirely endogenous in origin, *i.e.* they are due to some developmental abnormality

SYMPTOMATOLOGY

In the severe forms of diabetes the face is often wasted in appearance, the wrinkles and naso-labial folds are well marked, and the expression is often anxious or sad. But the face and lips are not anæmic, or not markedly anæmic, unless some complication should be present. In the mild forms of the disease, however, the facial expression is not characteristic.

In severe forms of diabetes *wasting* is a prominent symptom, especially in young persons, whilst in elderly patients mild diabetes is often associated with *obesity*.

ONSET.—In many cases *thirst* and *diuresis* are the first symptoms noticed; sometimes the earliest sign is troublesome *cramps* in the calf muscles at night. In other cases the patient first seeks medical advice on account of *wasting* and *increasing weakness*, or on account of one of the complications (gangrene, carbuncle, cataract, eczema of the genital organs, etc.). Occasionally the patient states very definitely that the thirst commenced suddenly on a certain day, and at a certain hour. Sometimes a very slight glycosuria, unaccompanied by thirst, diuresis, or other symptoms has preceded the true diabetes for months or years; but certainly this is not always the case.

THE URINE.—The *quantity* of urine is increased (often 150 to 300 ounces in the 24 hours); but in many of the milder cases the increase may only be slight (60-100 ounces daily).

The amount of urine is about equal to the fluid taken. It is reduced by nitrogenous diet and by intercurrent disease, and often diminishes before a fatal termination. The *colour* of the urine is very pale, generally light-yellow or straw-coloured; often, but not always, it has a *greenish*-yellow tint. But when the amount of urine is not increased, in the mild forms, the colour may be normal.

A mucous cloud, when present in diabetic urine, is often seen, not at the bottom, but at the upper part of the urine glass. Diabetic urine is usually bright and clear, but sometimes in female patients it is turbid from the presence of pus and epithelial cells. The *smell* is often sweet or aromatic; and in severe cases, especially just before the onset of coma, the urine has a peculiar "chloroform" smell, usually attributed to the presence of acetone. The taste of diabetic urine is sweet. The *reaction* is nearly always acid, and often markedly acid. The *specific gravity* is increased (up to 1030, 1045, or higher). A small quantity of sugar may be found, however, in urine of a normal or low sp. gr.

The *presence of sugar* is of course the most important change in the urine in diabetes. Sometimes before the patient has been aware of the nature of the disease he has been struck by the fact that flies have been attracted to his urine; also, if a drop of it has fallen on to his boot, or any adjacent object, and has been allowed to dry, he has observed that a salt-like deposit has been left behind. The sugar present in diabetic urine is grape-sugar (glucose), and its amount varies according to the nature of the case from 0·5 to 6, 8, or 12 per cent. The daily amount excreted may be 3000 to 4000 grains, or more. (For sugar tests see articles on "Glycosuria" and "Urine Testing.") The sugar increases in mild cases after food, and diminishes during fasting, and hence the excretion is less during the night. In very mild cases sugar may be absent in the night urine (passed before breakfast), whilst it is abundant in the day urine. The sugar excretion is increased by starchy and saccharine foods and diminished by nitrogenous diet. In mild cases, when carbohydrates are withdrawn from the diet, the sugar disappears from the urine, but returns when carbohydrates are taken again. In the mildest cases simple restriction of the carbohydrates is sufficient to cause the glycosuria to disappear. These are points of practical importance in urine testing in mild cases. In severe cases of diabetes sugar is present in the urine in spite of the withdrawal of all carbohydrate food, and may be present during fasting.

Sugar is the carbohydrate in the diet which causes glucose to be eliminated in the greatest quantity in the urine, whilst starch and other carbohydrates are less injurious. Grape-sugar is most injurious; fruit-sugar (lævulose) is only about half as injurious as grape-sugar; milk-sugar and cane-sugar stand midway between grape-sugar and lævulose. Fats never increase the sugar excretion, and alcohol in moderate quantity has no effect.

Muscular exercise diminishes the sugar excretion in well-nourished patients suffering from a mild form or early stage of the disease. But when the affection is severe, and the patient wasted, exercise increases the sugar excretion.

The sugar excretion is often *diminished* or *arrested* by intercurrent affections.

The excretion of *urea* is increased owing to the excess of nitrogenous food taken by the diabetic patient; only in a few cases is the increased urea excretion due to the destruction of the albumen of the body (Seegen).

Not infrequently there is a small deposit of uric acid crystals at the bottom of the urine glass in mild cases of diabetes.

The excretion of *ammonia* is increased in some cases, but not in all. In diabetes coma it is greatly increased. Sometimes there is an abundant deposit of oxalate of lime.

At the early stage of the disease *albuminuria* is usually absent, but it frequently appears at a late stage of the disease. Thus in 100 cases the writer found albumen present in 30 when the patients first came under observation; but in some of the cases in which albuminuria was absent at first it developed later, so that finally it was present in 44 per cent. The albuminuria is usually very slight, and not associated with nephritis or any gross lesion of the kidneys; but in a few cases the albuminuria is abundant, and there are signs of parenchymatous or interstitial nephritis. In only 4 out of the 100 cases of diabetes just mentioned was the amount of albumen large. In diabetic coma albumen and casts are nearly always present in the urine; but otherwise the albuminuria is not accompanied by casts, except in the rare case when there are indications of actual nephritis.

When inflammation of the prepuce or of the vulva is present as a complication, the urine, when recently passed, often contains fungus spores and mycelia along with a few pus cells. In diabetic females the urine is not infrequently slightly turbid, owing to the presence of pus and epithelial cells.

In the severe forms of diabetes, when a solution of perchloride of iron is added to the urine, there is at first a turbidity owing to the precipitate of phosphates, but on adding more of the perchloride solution a dark brownish-red coloration is often obtained. This coloration is known as *Gerhardt's reaction*, and is usually attributed to diacetic acid; by some it is attributed not to this substance, but to other closely-allied compound. This reaction is an indication of a severe form of the disease; it is usually present in diabetic coma; but in mild forms of the disease it is absent. A similar reaction is occasionally met with in febrile diseases and several other affections. Also the urine of patients who are taking salol, salicylic acid and salicylate of soda gives a dark brownish-red coloration with perchloride of iron; but the colour has more of a violet or purple tint than that obtained in severe cases of diabetes. The urine of patients taking antipyrin, salipyrin, and several other drugs also gives a brownish-red coloration with perchloride of iron. When the urine of a diabetic patient gives a reaction with perchloride of iron, often acetone is also present. It may be detected by Legal's test. To several cc. of urine a few drops of a concentrated, freshly-made solution of nitro-prússide of sodium are added. The mixture is made alkaline with liquor potassæ, and then acetic acid is added when a violet-red coloration is obtained. Another method is to distil about half a litre of urine; to the distillate a few drops of a solution of iodine in iodide of potassium, and a few drops of caustic potash are added. When acetone is present a precipitate of iodoform occurs. It is yellowish in colour, and has the characteristic iodoform smell; under the microscope it presents hexagonal plates or stars. It has been shown that acetonuria may be produced in healthy persons by a diet of nitrogenous and fatty food, free from carbohydrates: but on the addition of carbohydrates the acetonuria disappears (Hirschfeld, Rosenfeld, and others). A high degree of acetonuria or an increase of the acetone excretion is regarded by Hirschfeld as an indication of approaching coma.

In the severe forms of diabetes β-oxybutyric acid is often found in the urine.

THE BLOOD.—The most important change in the blood is the excess of sugar present. A minute quantity of sugar is present in normal blood (0·6 to 1·0, or a little more than 1·0 per 1000, Pavy; average in ten healthy men, 1·7 per 1000, Seegen). In diabetes mellitus the amount of sugar in the blood is usually greatly increased, and may reach 2·7 to 5·7 per 1000.

The writer has discovered a simple method of distinguishing diabetic blood from non-diabetic blood by its reaction with a solution of *methylene blue.* This method is extremely sensitive, and it is only necessary to examine a *drop* of blood obtained by pricking the finger. On heating a drop of diabetic blood and an alkaline solution of methylene blue in *certain* proportions the blue colour of the solution is removed, whilst if non-diabetic blood be used in place of diabetic blood (in the *same proportion*) the solution retains its blue colour.

The test may be performed as follows :—A small *narrow* test-tube is well cleaned, and a drop of water (40 *cubic millimeters*) placed at the bottom. It is important to use a narrow test-tube so that the upper surface of the fluid with which the air comes in contact may be as small as possible. To measure the quantity of blood the capillary tube of a Gowers' hæmoglobinometer which is graduated for 20 *cubic millimeters* may be used. The tip of one of the patient's fingers is cleaned and dried, and then pricked. When a large drop of blood has escaped it is sucked up into the capillary tube. Twenty *cubic millimeters* of blood are taken from the finger. The blood is blown gently into the water at the bottom of the small test-tube. Then 1 *cubic centimeter* (*i.e.* 1000 cubic millimeters) of a watery solution of methylene blue is added. The strength of the methylene blue solution is 1 in 6000. (To measure the methylene blue solution the 1 cc. pipette tube of Southall's ureometer may be used.) To the mixture of blood and methylene blue in the test-tube finally 40 *cubic millimeters* of liquor potassæ (B.P.) are added.

The fluids in the tube are then well mixed by shaking. As a control experiment a second test-tube of similar size is taken, and into this is placed the same quantity of non-diabetic blood with the same proportion of water, methylene blue, and liquor potassæ.

The fluid in each tube has a fairly deep-blue colour. Both small tubes are then placed in a beaker, capsule, or very wide test-tube containing water. Heat is applied by a spirit-lamp until the water boils. It is allowed to continue boiling for about four minutes. By the end of this time the fluid in the tube containing the diabetic blood changes its colour from a fairly deep-blue to a dirty pale yellow (almost the colour of normal urine), whilst the fluid in the tube containing the non-diabetic blood remains blue. Occasionally it becomes bluish-green, sometimes violet; but it never loses its blue colour. The tubes should be kept quite still whilst in the water-bath, as by shaking the decolorised methylene blue is apt to be oxidised by the oxygen of the atmosphere, and a blue tint may then return to the fluid. This is the reason why it is necessary to empty a water-bath, since if the test-tubes be heated directly over a spirit lamp it is difficult to avoid shaking the fluid.

A milky appearance of diabetic blood has occasionally been observed on post-mortem examination. In other cases the blood has had a pink colour, and on standing a milky or cream-like serum has separated on the surface. This cream-like condition of the serum has been shown by microscopical and chemical examination to be due to the presence of fat globules. Analysis of the blood has sometimes shown that the percentage of fat has been increased.

SYMPTOMS, COMPLICATIONS, AND PATHOLOGICAL CHANGES IN CONNECTION WITH THE VARIOUS SYSTEMS.—Besides the changes in the urine other prominent symptoms are—thirst, increased appetite, great weakness, and emaciation in the severe forms of the disease, a harsh dry skin, often a red, raw-looking tongue. The temperature is normal or subnormal except when complications occur.

ALIMENTARY CANAL, LIVER, AND PANCREAS.—The saliva is usually scanty and the mouth dry. The gums are often inflamed, spongy, and swollen. The teeth are often carious, and become loose and fall out. Thirst is one of the most characteristic symptoms of the disease, and often enormous quantities of fluid are taken: but in very mild cases the thirst is slight or absent; and also in severe cases during the last few days of life the thirst often diminishes or ceases, when phthisis or other complications have developed. The appetite is generally increased greatly, but this is not such a constant symptom as thirst. In certain mild cases the appetite is not increased; also in severe cases at a late stage the appetite fails.

Diabetic patients very frequently suffer from constipation. In chronic cases diarrhœa is occasionally present.

Pathological examination does not reveal any characteristic change in the stomach or intestines. Occasionally tuberculous ulceration of the intestines has been met with. The pathological changes in the liver and pancreas have been already referred to.

THE LUNGS.—The most frequent pulmonary complication is tubercular phthisis. It occurs more frequently in young diabetics, and in the severe forms of the disease amongst the poor. In elderly diabetics, especially in the obese or gouty, tuberculosis is much less frequent.

In 100 consecutive cases (chiefly hospital patients) in Manchester there were evidences of phthisis in 29.

Next to coma, tuberculous phthisis is the most common termination of diabetes. In half the cases of diabetes in which the writer has seen or made an autopsy, tuberculous disease of the lungs (extensive or slight) has been present. The phthisis of diabetic patients is usually tuberculous, and tubercle bacilli are present in the sputum.

Phthisis in diabetic subjects generally runs a comparatively latent course, and the pathological changes are more advanced than is suspected from the clinical signs. Cough and expectoration are often slight. The temperature is not much above normal, and hæmoptysis is very rare. But to these general statements there are exceptions. When the lung changes become advanced the diabetic symptoms gradually subside, and finally the sugar may disappear from the urine.

Pathologically diabetic phthisis is usually due to a chronic caseous tuberculous broncho-pneumonia, it generally runs a rapid course, caseation soon occurs, and the diseased parts break down and cavities are formed. There is no tendency to cicatrisation.

Occasionally a *chronic pneumonic* (non-tuberculous) phthisis is met with in diabetic subjects.

Gangrene of the lungs occasionally occur as a complication; in such cases the characteristic offensive smell of the sputum may be absent.

Broncho-pneumonia and acute croupous pneumonia are rare complications.

THE HEART.—Usually there are no signs of cardiac disease, especially in the severe forms of diabetes. (Heart normal on clinical examination in 93 out of 100 cases examined by the writer.) In the later stages of the disease the heart's action and the heart sounds are often feeble.

Mayer of Carlsbad has found cardiac enlargement in 21·6 per cent of the cases (but probably a considerable proportion of his patients would be elderly persons suffering from a mild form of the disease).

Occasionally diabetes terminates with symptoms of cardiac failure or collapse (see "Diabetic Coma," p. 489).

The pulse is usually normal, but in elderly persons often signs of arteriosclerosis and high tension are met with: and sometimes the pulse is large, hard, and of increased tension in diabetics under middle life, even when there are no evidences of kidney disease.

Pathologically in severe forms of the disease the heart usually presents no macroscopical change except general atrophy; or the heart muscle is often soft and flabby. Israel, however, found cardiac hypertrophy (generally associated with hypertrophy of the kidneys) in 10 per cent of the cases of diabetes examined pathologically at the Berlin Pathological Institute.

Sometimes fatty or glycogenic degeneration of the cardiac muscle is found on microscopical examination.

THE KIDNEYS.—Albumen, usually in small quantities or mere traces, is often present in the urine of diabetic patients (44 per cent of cases). It occurs especially at the late stages of the disease, and is not associated with nephritis. It has been attributed to excess of nitrogenous food, to catarrh of the bladder, and occasionally it is due to the mixture of a little pus with the urine owing to the balanitis in the male, or eczema of the vulva in the female. In a few cases the albumen is large in amount, and is associated with signs of nephritis, parenchymatous or interstitial (4 out of the 44 cases of albuminuria just mentioned).

On pathological examination, hypertrophy of the kidneys is not uncommonly found. Occasionally there are signs of interstitial, parenchymatous or diffuse (parenchymatous with interstitial) nephritis, and in a few cases tubercle of the kidneys. Microscopically changes in the renal epithelium are often present, and the following have been described:—hyaline degeneration (Armanni); necrosis of epithelium (Ebstein), fatty degeneration (Fichtner); glycogenic degeneration (Ehrlich and Frerichs). The latter change is met with chiefly in the cells of Henle's loop.

THE SKIN.—In severe cases the skin is usually dry and rough, but in the milder cases it is generally normal. Pruritus is sometimes troublesome, it is usually local, rarely general. Pruritus is most common about the genital organs especially in females, and sometimes it is the symptoms for which advice is first sought. Pruritus of the vulva is produced by the irritation of the saccharine urine, it is often followed by erythema and eczema. In the male pruritus of the glans penis is due to the same cause, and is often followed by erythema and balanitis, and the prepuce may become œdematous.

Boils and carbuncles sometimes occur; the latter are most frequently met with on the neck. In patients over 45 gangrene sometimes occurs. It may be moist or dry, and is frequently associated with arterio-sclerosis. It occurs most frequently in the foot, and is often excited by some slight wound or injury to the skin. Gangrene may follow cellutitis in other parts.

Perforating ulcers on the feet resembling those seen in locomotor ataxia are occasionally met with in diabetics over middle age.

Anasarca of the feet and sometimes of the hands, without albuminuria or signs of cardiac failure, is an occasional complication of diabetes.

Xanthoma diabeticorum is one of the rarest skin affections in diabetes.

Other skin affections are occasionally met with, and recently a form of diabetes with bronzing of the skin has been described (*diabète bronzé*).

AFFECTIONS OF THE EYE.—Defects of vision are not uncommon in diabetes. The most important affection is cataract (9 per cent of cases). It is usually bi-lateral, and occurs in young patients as well as in the aged. It is generally of the soft variety but not invariably.

Defects of vision are also caused by paresis of accommodation, short-sightedness developing late in life, vitreous opacities, retinitis, and amblyopia. Diabetic retinitis is very rare (seven cases in 100). It only occurs in patients over the age of 40. In one form of diabetic retinitis the changes consist of small punctiform hæmorrhages scattered over the retina. In another form the changes consist chiefly of small white patches chiefly near the centre of the retina. These white patches have a "curdy" appearance, and are sometimes clustered together in the form of a semicircle or incomplete circle surrounding the yellow spot; they are never grouped in a star-shaped form radiating from the yellow spot as in albuminuric retinitis.

Occasionally diabetic patients suffer from amblyopia with defect of vision chiefly in the centre of the field and with central scotomata for colours. Ophthalmoscopic examination reveals no changes in the disc or retina, and the condition resembles tobacco amblyopia.

SEXUAL FUNCTIONS.—In the male, diminution or loss of sexual power is not infrequent. In females the sexual desire is said to be diminished greatly in severe cases, whilst in the mild forms of the disease in elderly women it is said to be increased. Amenorrhœa sometimes occurs at an early stage. Pregnant diabetic women often abort, and during pregnancy and the puerperal state the disease often advances markedly.

NERVOUS SYSTEM.—Mental dulness and drowsiness are frequent. Often the patient is melancholic and depressed, but as a rule the intellect remains clear up to the last. The writer has recorded two cases in which changes were present in the posterior columns of the cord in diabetic patients. In both cases the changes were seen clearly with the naked eye. After hardening in Müller's fluid the degenerated tracts were paler in colour than the rest of the white matter; in both cases they occupied Goll's columns in the cervical region; they extended into Burdach's columns in the dorsal region in one case, in the lumbar region in the other. Microscopically the changes were much less distinct, and consisted in slight degeneration of the fibres of the posterior columns. Probably they were the result of the diabetic blood condition. Similar changes have been recorded by several observers. *Cramps* in the calves of the legs at night are common in diabetes, and are sometimes amongst the earliest symptoms of the disease. The patient may also complain of gnawing pains in the legs, which may be so tender that he cannot bear one leg to lie over the other in bed. The legs may feel numb, and the knee-jerks in such cases are often absent. These symptoms are probably due to slight peripheral neuritis. Occasionally, but very rarely, there are well-marked symptoms of peripheral neuritis—the feet being "dropped" and the legs paralysed, and sensory symptoms being present as in ordinary alcoholic neuritis. Cases are on record of monoplegia and of paralysis of single groups of muscles which are probably of neuritic origin.

A small number of cases have been published in which pathological examination has verified the diagnosis of peripheral neuritis (parenchymatous form). In most of the cases recorded the neuritis has occurred in patients over the age of 50, and this complication does not bear any relation to the amount of sugar in the urine.

The *knee-jerks* are not infrequently lost in diabetic patients, and in such cases there are sometimes slight signs of neuritis (gnawing pains in the legs, tenderness, and cramps in the calf muscles); very rarely there are marked signs of *peripheral neuritis.* But frequently when the knee-jerks are lost there are no other indications of neuritis, and pathological examination may reveal no changes in the peripheral nerves. Amongst 100 diabetic hospital patients the writer found the knee-jerks both lost in 49, both present in 45, one present, one absent in 6. Most of these patients suffered from a very severe form of the disease, with great wasting. In private practice, where most of the mild forms and early cases are met with, the knee-jerks are only lost in a small proportion of cases. The knee-jerks when present at an early period are frequently lost later. During the last few days of life, the writer found the knee-jerks lost in 73 per cent of hospital cases, and they were lost in 20 out of 23 cases of diabetic coma. In the severe forms of the disease, when the knee-jerks have been absent, the writer has usually found the wrist-jerks also absent. But the superficial reflexes — plantar, abdominal, and epigastric — are generally readily obtained or increased, even when the knee and wrist jerks are absent.

DIABETIC COMA.— The most frequent termination of diabetes is in coma, which is not dependent on any gross lesion of the organs. When advanced phthisis is present as a complication, usually coma does not occur, but to this rule there are exceptions.

Diabetic coma may occur in all forms of the disease and at all ages, but it is especially common in young patients and in the severe forms. Coma may develop at a very early date after the onset of diabetes, especially in young people (few weeks or months), or it may only occur after the disease has been present for years. Coma has frequently developed directly after a long railway journey. Great muscular exertion, great mental worry and anxiety are also exciting causes. A sudden change of diet and a very rigid diet are thought by many (Ebstein, Naunyn, Schmitz, Grube, etc.) to favour the development of diabetic coma when the patient is suffering from a severe form of the disease, and when the urine gives a marked reaction with perchloride of iron. Prolonged constipation is probably a predisposing cause, and numerous cases are on record in which various complications and also surgical operations have appeared to be exciting causes.

The symptoms of diabetic coma often commence with lassitude, epigastric pain, nausea, and occasional vomiting. In other cases shortness of breath is the earliest symptom, and it has occasionally preceded other symptoms for several days. Headache may occur at the onset. Often the patient is anxious, restless, or excited at first, then drowsiness gradually develops and passes into coma. The pulse becomes rapid and feeble, and Lépine regards rapidity of the pulse as an important early sign of commencing coma. Dyspnœa is a prominent feature in the majority of cases, and the breathing has a peculiar panting or sighing character; the number of respirations per minute is often only slightly increased or even normal, and the dyspnœa is often indicated by deep inspiration and deep expiration, rather than by much increase in the frequency of respiration. This peculiar dyspnœa is described by Kussmaul as air-hunger. The bowels are generally constipated. The skin becomes pale and cold; and in many cases there is slight cyanosis of the nose, lips, ears, hands, and feet. The temperature is generally subnormal, 95°-97° F.; in a few cases it rises at the last to 102°-104° F. The breath has usually a peculiar smell, somewhat like chloroform, and acetone has been found in the expired air. This smell is often detected for some time before the onset of coma.

The acidity of the urine is increased, but the quantity of urine and the amount of sugar excreted usually diminish with the onset of comatose symptoms. The urine, like the breath, has a peculiar "chloroform" smell (acetone). A trace or small quantity of albumen is usually present (according to some writers invariably present) in the urine in the common variety of diabetic coma. Sometimes the urine is free from albumen up to the onset of comatose symptoms; but in other cases a trace of albumen is present for some time before coma develops. In 20 consecutive cases of diabetic coma the writer found a trace or small quantity of albumen in the urine in every case. According to Külz, casts are always present in the urine in the common form of diabetic coma, and he believes the appearance of casts to be a valuable premonitory sign of the onset of coma. In 17 consecutive cases of diabetic coma, the writer found them always present. When a small amount of a dense yellowish-white deposit appears exactly at the bottom of the urine glass, and when this deposit consists of casts, he believes that coma nearly always follows. The casts in diabetic coma are finely granular or hyaline, and are present in enormous numbers. The urine in diabetic coma usually gives a dark brownish-red coloration with perchloride of iron (Gerhardt's reaction). Acetone can also be detected. The amount of ammonia is much increased, and according to Stadelmann β-oxybutyric acid is always present. The alkalinity of the blood is said to be much decreased. The blood always decolorises an alkaline methylene blue solution as already described. The knee-jerks are usually absent (absent in 20 out of 23 cases).

The coma often becomes complete; in other cases the patient, though semi-comatose, can be roused almost up to the last. When once well-marked symptoms of coma have developed, death occurs within forty-eight hours.

The description just given is that of the common form of diabetic coma (Kussmaul's variety), but there are two rare sub-varieties. (1) The *alcoholic form.* At the onset the patient is very excited, and behaves like a drunken man. The pulse is quick, coma develops, and soon terminates fatally. Such cases have been mistaken for alcoholic intoxication. The urine gives the reaction with perchloride of iron, the breath has the "acetone" smell, but dyspnœa is absent or slight. (2) *Diabetic collapse* (described by Dreschfeld and Frerichs). The patient suddenly becomes drowsy, the extremities and face cold and livid, the pulse quick and small, and coma develops. The temperature sinks subnormal, the breath has not the "acetone" smell, and the urine does not contain acetone or diacetic acid. Dreschfeld points out that this form of coma occurs chiefly in patients over 40, and often they are well nourished or stout. Dreschfeld and Frerichs believe that the symptoms are due to cardiac failure owing to degeneration of the cardiac muscle.

The Diagnosis of diabetic coma (in its common form) is easy, especially if the patient is known to have been suffering from diabetes for some time. If the patient be seen for the first time in the comatose state, then the diagnosis between diabetic coma and other forms of coma has to be considered. If the urine can be obtained, the presence of a large quantity of sugar, along with a high specific gravity, is sufficient to exclude most of the other causes of coma. The "acetone" smell of the breath, the perchloride of iron reaction in the urine, and the dyspnœa are also important indications in favour of diabetic coma. If the urine cannot be obtained, or if there should be any doubt as to the nature of the case, the methylene blue reaction obtained by examination of a drop of the patient's blood will definitely settle the diagnosis.

In coma from fractured skull and other cerebral conditions occasionally the urine contains sugar, but the quantity is only small, the specific gravity is not high, there is no perchloride of iron reaction, there is no "acetone" smell of the breath and the urine, the peculiar dyspnœa is absent, and the pulse is often slow or normal; whilst in diabetic coma of the usual form the pulse is rapid, and dyspnœa is a prominent symptom. Symptoms resembling those of Kussmaul's diabetic coma are said to occur occasionally in other affections (cancer of the stomach, anæmia, kidney disease), but in such cases (as well as in the alcoholic and cardiac forms of diabetic coma) the diagnosis can be made by the urine examination or by the methylene blue reaction of a drop of the blood.

The pathological changes met with in patients dying of diabetic coma are not characteristic. The most constant are those in the renal epithelium already mentioned. In the variety of coma described as diabetic collapse, it is very probable that the cause is cardiac failure owing to degeneration of the heart muscle. In the common variety of diabetic coma, fat embolism has been suggested as a cause in certain cases. According to Schmitz, the symptoms are due to a ptomaine formed in the intestine owing to obstinate constipation. The symptoms certainly point to the action of some toxic substance. Acetone and diacetic acid have been suggested as the poison, but both substances can be given in large doses without producing coma (Dreschfeld and others). There is considerable evidence in favour of intoxication by some organic acid (Stadelmann), and β-oxybutyric acid or crotonic acid are thought by some to be the toxic substances. As already pointed out, the urine nearly always contains casts and a small quantity of albumen in diabetic coma, and the renal epithelium frequently presents degenerative changes. Hence it appears probable that there is impairment of the renal functions. V. Harley has shown that symptoms resembling diabetic coma can be produced in animals by injecting grape-sugar into the jugular vein and preventing its elimination by ligaturing the ureters. Now in diabetic coma the sugar elimination and urine secretion usually diminish, and from a consideration of facts mentioned it appears not improbable that coma is due to poisoning owing to the kidneys failing to eliminate certain substances produced in the diabetic organism.

Forms of Diabetes and Glycosuria.—There are two chief forms besides several sub-varieties. In the *severe* form the sugar excretion is great and does not cease when carbohydrates are excluded from the diet. There is often much wasting. This form is most common in patients under middle age. In children and young persons it sometimes runs a very rapid course (a few months or even less). In the *mild* forms of diabetes the symptoms and sugar excretion are not so marked. The sugar disappears from the urine on withdrawing the carbohydrates from the food. The patients are often above middle age, and they are not infrequently stout or gouty. The night urine contains less sugar than the day urine, or it may be free from sugar. The course is often chronic. (This form is sometimes spoken of as chronic glycosuria.) There are also *transitional* and *intermediate* varieties, and the mild form may pass into the severe. In some of the mildest forms thirst, diuresis, and other symptoms are absent, and the only signs of disease are made out by examination of the urine, which contains sugar and has a high specific gravity (*diabetes decipiens*). In another mild form the glycosuria and other symptoms disappear from time to time (*intermittent* diabetes). It has already been pointed out that glycosuria may be secondary to a number of affections (*symptomatic* glycosuria—see article "Glycosuria"). Hanot, Chauffard, and others have drawn attention to a rare disease characterised by the association of symptoms of diabetes mellitus with bronzed pigmentation of the skin, and frequently with hypertrophic cirrhosis of the liver (*diabète bronzé*).

TERMINATION.—The severe forms always terminate fatally. The duration may be a few years (3-5); often it is shorter; occasionally it is a few months, or even less. In the mild cases life may be prolonged for many years (10 or 20). In the mild cases by restricted or rigid diet the symptoms may disappear entirely, but they usually return when an ordinary diet is taken.

In rare cases diabetes is associated with symptoms of nephritis (parenchymatous or interstitial), and the diabetic symptoms may gradually subside whilst those of nephritis remain. Also occasionally (though very rarely) the symptoms of diabetes mellitus give place to those of diabetic insipidus.

The fatal termination in diabetes mellitus is most frequently by diabetic coma. The next most frequent termination is by pulmonary phthisis. Other causes of death are carbuncle, gangrene, etc.

THE PROGNOSIS depends chiefly on the form of the disease and the age of the patient. In the mild forms, especially if the sugar excretion can be checked by a rigid diet, and if the patient be over middle age, the prognosis is fairly favourable. But it is very grave in young persons and in the severe forms of the disease. Other unfavourable indications are marked wasting, the occurrence of pulmonary tuberculosis, a family history of the disease, unfavourable conditions of life, Gerhardt's perchloride of iron reaction in the urine, signs of commencing coma, onset of gangrene.

Favourable indications in addition to those already mentioned are the association of obesity or gout, long duration of the disease without much wasting, favourable conditions of life, in the female onset about the climacteric period.

THE DIAGNOSIS of diabetes mellitus is usually easy. The disease may be overlooked, however, and the patient treated simply for one of the complications. It is important to remember that occasionally the patient complains chiefly of weakness and wasting. It is important not to mistake a temporary and mild glycosuria for true diabetes.

PATHOLOGICAL ANATOMY.—Numerous pathological changes have been described in diabetes, yet, strictly speaking, the disease has no definite pathological anatomy. The changes met with are usually due to complications or are secondary in nature, and it would scarcely be possible for a pathologist to diagnose diabetes without a clinical history or the chemical examination of the urine or blood. For this reason the changes found post-mortem in the various organs have been already described in considering the relation of diabetes to disease of the nervous system, liver, and pancreas, and in the description of the complications. The condition of the blood has been described under the symptomatology.

Pathogenesis.—The true nature of the disease remains obscure. The theories as to its exact origin are numerous, but none is altogether satisfactory. Sugar appears in the urine because there is an excess of sugar in the blood, and the symptoms of the disease are due to the same cause.

The cause of the excess of sugar in the blood has been attributed to (1) an excessive formation of sugar in the system ; (2) a diminished sugar destruction ; (3) by some writers it is believed, that in certain cases there is an excessive sugar formation, in others a diminished sugar destruction. Bunge has given good reasons for rejecting the view of excessive formation as regards the origin of the sugar from articles of food. Kaufmann, on the other hand, has brought forward experimental evidence in favour of increased sugar formation. He isolated the liver by trying all its vessels in a healthy dog, and also in another dog which had been rendered diabetic by extirpation of the pancreas. The sugar in the blood gradually became diminished during its circulation in various parts of the body, but the blood lost the same proportion of sugar both in the healthy and in the diabetic animal. Hence, he concludes that sugar destruction was the same in each.

Many authors believe that diabetes in man is usually due to diminished sugar destruction.

In the mild forms of diabetes removal of carbohydrates from the diet causes the glycosuria to cease. In such cases the sugar appears to be derived in some way, directly or indirectly, from the carbohydrates of the food. Seegen believes that in the mild form (which he terms the "hepatogenic") the liver cells are unable to assimilate the carbohydrates in a normal manner. But pathological anatomy has failed to reveal any definite changes in the liver associated with diabetes.

According to Pavy, the two lines of defence—intestinal villi and the liver—are inadequate to accomplish the function of synthesising the carbohydrates. Hence the latter reach the general circulation in excessive quantity and appear as sugar in the urine.

In the severe forms of diabetes the sugar in the urine is evidently not dependent simply on the carbohydrates of the food. Since the glycosuria persists when the diet consists only of fat and nitrogenous substances, and persists even when no food is taken, Pavy believes that in these severe forms the sugar is derived not only from the food but also from the tissues. He believes that the proteids of the body have a glucoside constitution, and that in diabetes of the severe form a carbohydrate is cleaved off from these proteids by the action of some ferment which he supposes to be present in the system.

According to Seegen, in the severe forms the cells and tissues of the organisms have lost their function of destroying the sugar in the blood.

Limited space forbids a discussion of the subject, but whatever view we take of the sugar formation or destruction the question finally arises—*What is the cause of the abnormality in diabetes?*

Pavy believes that as regards the liver there is a vasomotor paralysis and dilatation of the small arterioles owing to some change in the nervous system. As a result the blood in the liver is in a hyper-oxygenated state, and this favours the passage of carbohydrates into glucose. But Seegen points out that Pavy was able to produce diabetes experimentally by injury to the nervous system after ligature of the hepatic artery, and he asks therefore how vasomotor paralysis can be regarded as the explanation.

It has been already shown that there is strong evidence that some change in the nervous system is the starting-point of the disease in many cases.

Also experiments on animals and pathological observations render it very probable that, in some cases of diabetes, pancreatic changes are the cause of the disease.

It has also been pointed out that atheroma might act as the starting-point of diabetes by producing changes in the nervous system or in the pancreas, and there are a few cases on record which afford strong evidence of diabetes being the result of pancreatic changes which were secondary to atheroma.

Diabetes has been attributed by Bunge and others to pathological chemical changes commencing in the muscles.

The opinion appears to be gradually gaining ground that diabetes is not a pathological entity, but rather a group of symptoms which may be produced by the various morbid changes already suggested. Possibly it is sometimes due to an endogenous or inherited morbid condition, and possibly to other causes.

TREATMENT

The results of treatment in the mild forms are good, in the severe forms very unsatisfactory. After a diagnosis has been made, before commencing treatment, the weight should be taken, complications noted, the amount of sugar estimated, and the urine tested with perchloride of iron. Then a rigid diet (practically free from carbohydrates) should be prescribed in order to ascertain whether the sugar excretion can be checked thereby. If this should occur the case belongs to the mild form of the disease, and a little carbohydrate food in the form of bread is then allowed and gradually increased (if necessary) until the glycosuria returns. The amount of bread which can be taken before the return of the glycosuria is thus an indication of the quantity of carbohydrate food the patient can tolerate. If a diet free from carbohydrates does not cause the sugar to disappear from the urine, then the patient is suffering from a severe form of the disease. If there should be great wasting, and especially if the urine should give a marked reaction with perchloride of iron, it is not advisable to keep the patient long on this very rigid diet.

Many authors think it is important in the very severe forms of the disease that the rigid test diet should not be commenced suddenly, as by such a procedure there is a possibility of producing diabetic coma (Ebstein, Naunyn, and others). A few days may be allowed to elapse in changing from a mixed to a rigid diet. Potatoes may be excluded first, then bread, and afterwards all other carbohydrates. This plan appears to the writer to be important.

Having determined the effect of diet and the form of the disease in any case, directions must be given for the future DIETETIC TREATMENT—(1) as to the *nature* of the food; (2) as to its *quantity*. The treatment must be modified in each case according to the form of the disease. A record of the weight and sugar excretion should be kept, and probably the former is the more important.

IN THE MILD FORMS OF THE DISEASE there are two classes of cases: (1) those in which the sugar excretion cease only when carbohydrates are excluded from the diet; (2) those in which it ceases when the carbohydrates food is simply diminished in quantity. In either case the diet which is sufficient to arrest the glycosuria should be continued for several weeks. It is often found that the condition of the patient is then much improved. In the first class of cases he is able to take a little carbohydrate; in the second class he is able to take an increased amount of carbohydrate without the glycosuria recurring. But in many of the first class of cases any addition of carbohydrates to the diet is followed by a return of the glycosuria. After a period of restriction, however, it is usually necessary to relax the diet a little, especially as regards bread, and to remain content, if by a moderately restricted diet we can limit the sugar excretion to 500 or 600 grains daily. If the patient should be very stout a reduction of the total quantity of food is often of great service, but if wasted this should not be attempted.

IN THE SEVERE FORMS OF THE DISEASE, when the most rigid diet fails to remove sugar from the urine, and when there is much wasting, and especially if the urine give a marked reaction with perchloride of iron, the opinion has been gradually gaining ground for some years that a very rigid diet is injurious. In such cases, after a short period of rigid dietary (in order to determine the form of the disease), a small amount of carbohydrate food must be allowed. The diet in these severe forms should consist of nitrogenous food, fatty food, and a small amount of carbohydrate food, chiefly in the form of bread; but saccharine food should be avoided, and the carbohydrate food should only be allowed in limited quantity. Fatty food is especially important, and should be given in large quantities, a little alcohol being also allowed to aid its digestion. Cream is of great service, and should be given freely; also milk may be allowed in moderate quantities. If coma appears to be threatening the diet should be less rigid, *i.e.* the amount of carbohydrate should be increased. According to Ebstein, the appearance of acetone and diacetic acid in the urine is an indication for diminishing the albumen and for increasing the carbohydrates.

ARTICLES OF DIET.—When a rigid diet is indicated (either for diagnosis or for treatment), the following articles should be sanctioned or forbidden:—

Sanctioned	*Forbidden*
Butchers' meat of all kinds (except liver), potted and preserved meats.	**Sugar, saccharine and farinaceous articles of food.**
Ham, tongue, bacon.	**Pastry and farinaceous puddings.**
Poultry, game.	
Fish (fresh, dried, and preserved), sardines, shrimps.	
Broths, animal soups, and jellies (prepared without the addition of saccharine or starchy materials).	**Rice, sago, arrowroot, tapioca, macaroni, vermicelli, semolina.** **Potatoes.**
Eggs, cheese, cream.	
Butter, suet, oils, and fats.	
Custard (without sugar).	
Reliable bread substitutes (gluten bread, almond and aleuronat cakes).	**Wheaten bread and biscuits.**
Green vegetables—mustard and cress, watercress, endive, lettuce, spinach, turnip-tops, cabbage, broccoli, Brussels sprouts, spring onions.	**Carrots, turnips, parsnips, beetroot, beans, peas, large onions.**
Cucumber.	**Liver.**
Mushrooms.	**Oysters, cockles, mussels, the "puddings" of crabs and lobsters.**
Pickles (cucumber, walnuts, and onions).	**Honey.**
Nuts (walnuts, almonds, filberts, hazel nuts, Brazil nuts), but not chestnuts.	**All sweet fruit and dried fruits.**

BEVERAGES

Sanctioned	*Forbidden*
Water, soda-water, and mineral waters.	**Port, Tokay, champagne, and sweet wines.**
Tea, coffee.	**Must, fruit juices and syrups.**
Dry sherry, claret, Burgundy, hock, Moselle, Ahr wines, most Rhine wines, Austrian and Hungarian table wines (all in moderate quantities, however).	**Sweet lemonade.** **Liqueurs.** **Beer, ale, porter, and stout.**
Brandy in small quantities.	**Rum and sweetened gin.** **Cocoa and chocolate.** **Milk in large quantities.**

Almost all kinds of animal food may be sanctioned, but liver, oysters, cockles, mussels, crabs, and lobsters should be avoided, because they contain a large amount of carbohydrate material.

With regard to milk caution is necessary, since it contains 4 per cent of milk-sugar, but it also contains albuminous bodies and fat, which are of great service to the diabetic. In some cases the addition of milk in considerable quantity to the diet of a diabetic patient does not increase the sugar excretion, *i.e.* the milk sugar is utilised in the system. In other cases the glycosuria is distinctly increased by the milk, but nevertheless such patients may gain weight. In the very severe forms of the disease milk is of great service, especially if the digestion is feeble, but in milder forms, if a rigid diet be desirable, milk should be excluded, unless it can be shown that it does not increase the glycosuria.

Cream contains less lactose than milk, but seven times the amount of fat. It may be taken freely by diabetic patients, and is most useful in the severe forms of the disease. By the following simple method an *artificial milk* can be prepared from cream:—To about a pint of water placed in a pot or glass measure four tablespoonfuls of fresh cream are added and well mixed. The mixture is allowed to stand for twelve hours. Most of the fatty matter of the cream floats to the top, and can be skimmed off with a teaspoon, and on examination it will be found almost free from sugar (the sugar originally present in the cream having been dissolved and distributed in the water). This fatty matter is then separated and placed in a glass and mixed with water. The white of an egg is added, and the mixture well stirred. A little salt and a trace of saccharine may be added. The mixture with a little practice may be made to taste almost like milk, and it may be taken freely by all diabetic patients.

Fats are the most valuable articles of diet for diabetic patients, especially in the severe forms of the disease, and may be allowed in large quantities. The most useful are butter, cream, bacon, cheese, eggs, suet. Cod liver oil is also of service. If fatty food should give rise to dyspepsia, a small amount of brandy and water, or other form of alcohol, taken after the meal, often aids the digestion.

Of the *carbohydrates*, starch is less injurious than sugar. Of the various kinds of sugar, glucose is most injurious. Milk-sugar and cane-sugar rank next. Lævulose is least injurious. Many observations have shown that in moderate quantities lævulose is utilised in the system, and does not increase the sugar excretion in mild forms of diabetes; but in large quantities, and in the severe forms of the disease, it is only partially utilised, and therefore the sugar excretion is increased. Saccharine and saxine may be used to sweeten articles of food in place of sugar.

When a very rigid diet is indicated, *fruit* should be excluded, on account of the sugar which it contains. But in many kinds of fruit a large portion of the sugar is lævulose, which, as just mentioned, can be utilised in small quantities in certain cases. Hence when a very rigid diet is not necessary, a very small quantity of those fruits which contain least sugar may be allowed.

Grapes, cherries, and other fruits which contain much sugar, as well as dates, figs, currants, raisins, and other dried fruit, should be forbidden. Nuts may be allowed freely, with the exception of chestnuts. As a rule green vegetables may be allowed, whilst white vegetables and root vegetables, which contain more carbohydrate, should be avoided. These have been already indicated.

Bread is the article of diet with respect to which there is the greatest difficulty. It contains 49 per cent of carbohydrates, and 2 per cent of sugar (König), and is thus unsuitable when a very rigid diet is desirable for diagnosis or treatment. In such cases it must be replaced by various bread substitutes. Unfortunately many of these are very unreliable, and contain either starch or sugar in considerable quantity. Before recommending any specimen it is important to try the effect of a drop of iodine and potassium iodide solution. If the specimen becomes deep blue black it contains a large quantity of starch, and is unreliable. Some bread substitutes contain sugar, and hence it is advisable to test for sugar by the fermentation test in an inverted test-tube.

In the most severe forms of the disease, when a very rigid diet is not desirable, it is best to allow a small amount of ordinary bread daily; in other cases, when for diagnosis or treatment a very rigid diet is necessary, bread should be excluded from the diet for a few weeks, and some reliable substitute employed. But after a month or two most patients find bread substitutes objectionable, and it is necessary and often desirable to allow a small quantity of ordinary bread. The following bread substitutes have been employed:—

(i.) *Gluten Bread.*—Good preparations of gluten flour are almost unaffected by iodine, and contain only 2 to 3 per cent of carbohydrate. The flour can be purchased and the bread prepared at the patient's house according to the directions supplied by various firms. With care a fairly palatable and reliable preparation may be obtained which will be of service as a bread substitute, and will cause a reduction of the sugar excretion. But many preparations are very unpalatable and unreliable. (ii.) *Bran cake* was recommended by Camplin many years ago, but is now seldom used. (iii.) *Soya biscuit* and bread have been largely used recently. They are prepared from soya beans, and some specimens contain only a small percentage of starch (3 to 6), but others contain as much as 30 or 45, and are therefore useless. (iv.) *Almond cakes* were recommended by Pavy long ago. They can be prepared at the patient's home from almond flour. About 4 ounces of almond flour are mixed into a paste with a little water and German yeast. The mixture is allowed to stand in a warm place for about twenty minutes. Then one egg, beaten up, and a little cream and water are added, and the whole is mixed into a paste, divided into cakes, and baked for fifteen to thirty minutes. (v.) *Cocoa-nut* cakes may be prepared in the same way by using dessicated cocoa-nut powder in place of almond flour. Both almond flour and cocoa-nut powder contain a small amount of sugar, which is destroyed by the action of the yeast used in making the cakes. Both of these cakes contain a large amount of fat, and hence a little alcohol is often necessary to aid their digestion. Almond flour and cocoa-nut powder may be used for the preparation of pudding.

(vi.) *Aleuronat* is a vegetable albuminous substance which contains only a very small percentage of carbohydrate, and which has been strongly recommended by Ebstein. The writer has found that palatable and reliable biscuits can be prepared by mixing aleuronat and cocoa-nut powder.

Two ounces of dessicated cocoa-nut powder are mixed with a little water and German yeast, so as to form a paste. This is kept in a warm place for half an hour, then 2 ounces of aleuronat, one egg (beaten up), and a small quantity of water, with a little saccharine, are added. The whole is mixed into a paste, which is spread out on a tin, divided into cakes, and baked for twenty or thirty minutes.

THE BEVERAGES which are free, or almost free, from carbohydrates may be allowed, and they have been already indicated. Alcohol does not increase the sugar excretion, and therefore alcoholic drinks which contain only a very small quantity of carbohydrate may be allowed in moderation. In the severe forms of the disease, alcohol is of service in enabling the patient to take large quantities of fatty food without dyspeptic troubles.

The following is a useful lemonade for the relief of thirst. Citric acid 10 grains, glycerine 4 drachms, water one pint. This may be taken in small quantities during the twenty-four hours; or a lemonade may be made from fresh lemons and sweetened with saccharine. Other acid drinks may be used. The largest quantity of fluid should be taken before, not after a meal.

THE MODE OF LIFE AND GENERAL HYGENIC CONDITIONS.—It is important to relieve the patient from mental worry and anxiety as much as possible. Considerable muscular exercise in the open air is of great service in some of the milder cases, and sometimes causes a diminution of the glycosuria, but in the severe forms it is injurious. Massage is of service in some cases (according to Grube in those associated with arterio-sclerosis).

Treatment by Alkaline Mineral Waters.—The spas of Carlsbad, Marienbad, Neuenahr, and Vichy are much frequented by diabetic patients on account of the supposed virtues of their mineral waters. The salts in the waters of Carlsbad and Marienbad consist chiefly of sodium sulphate and sodium bicarbonate; those in the waters of Neuenahr and Vichy chiefly of sodium bicarbonate. It is useless and often dangerous to send English patients suffering from the *severe* forms of diabetes to these spas. The long journey has not infrequently acted as an exciting cause of diabetic coma. Arterio-sclerosis is stated by Schmitz to be a contra-indication of the Neuenahr waters. It is certain, however, that patients suffering from the mild forms of the disease (chiefly those which are associated with obesity or gout) do often derive much benefit from a visit to these spas. But whether the results are due to the action of the waters or to the improved general conditions of life, more careful diet, and life in the open air, is a disputed point. (But from limited observations at Carlsbad and Neuenahr, the writer is inclined to attribute part of the good results obtained in the mild cases to the action of the waters.)

Medical Treatment.—It is certain that no drug hitherto tried has a definite curative action, but several have a beneficial effect.

Opium and its alkaloids have been long employed, and are probably more useful than other drugs. At first half a grain of opium may be given three times a day. The dose may be gradually increased to 2 or 3 or more grains of opium three times a day, as diabetic patients are very tolerant of the drug. Ralfe advised it to be given an hour after a meal, since at this time he believed it had a greater effect in restraining diuresis, and was less liable to cause dyspeptic symptoms. Some authors prefer the crude opium, others morphia, and others codeia. The last drug has been specially recommended by Pavy. It may be given in small doses at first, half a grain three times a day, and gradually increased to 2 or 3 grains. All the opium preparations are liable to cause troublesome constipation and dyspeptic symptoms, and therefore require watching, but probably codeia is less injurious in this respect.

Next to opium and its alkaloid, *alkalies* have been, perhaps, most frequently employed in the treatment of diabetes. The alkalies which are most often used are the bicarbonate of soda, and the citrate, acetate, carbonate or bicarbonate of potash.

Naunyn strongly advocates the use of sodium bicarbonate in large doses in severe cases of diabetes before the onset of symptoms of coma, and when there are indications of acid intoxication—such as great excretion of ammonia or marked reaction of the urine with perchloride of iron. So long as the perchloride of iron reaction is intense he advises that bicarbonate of soda (150 to 450 grains daily) should be given, and that it should be increased in amount if the perchloride reaction increases. Since adopting this treatment (twelve years ago) the number of cases of coma in his practice has diminished greatly.

Lithium salts (carbonate and citrate) have been much employed (often combined with arsenic). In cases associated with gout they may be of service.

Arsenic has been much prescribed in the treatment of diabetes, and in some of the milder cases it probably is of some service, when the dose is gradually increased (up to 10 m. of the liquor arsenicalis three times a day).

Jambul has been often prescribed in diabetes during recent years. Many observers have found it useless; others have obtained favourable results when the drug has been given in sufficiently large doses.

Sodium salicylate has been recommended by Ebstein, who prescribes it freely. Brunton and Ralfe think it is of most service in the glycosuria of gouty persons. Schmitz of Neunahr obtained better results with *salicylate of bismuth* than with any other drug in the mild forms of the disease (7½ grains twice a day).

Recently *uranium nitrate* has been recommended by West, and there is certainly some evidence in its favour.

Numerous other drugs have been recommended from time to time, but usually extended experience has shown them to be useless or of very slight value.

Cod liver oil or lipanin are to be recommended when the patient is much wasted.

TREATMENT OF COMPLICATIONS.—When irritation and eczema of the genital organs (vulva or prepuce) is a troublesome symptom, it is important that the external orifice of the urethra and surrounding parts should be dried with lint or absorbent cotton wool directly after each act of micturition. Boracic acid ointment, boracic acid lotion, or a solution of sodium hyposulphite (one in forty) may be applied. The treatment of other complications—boils, carbuncles, gangrene, phthisis, nephritis, etc.—is described elsewhere in the articles devoted to these subjects.

PREVENTION AND TREATMENT OF DIABETIC COMA.—In the severe forms of diabetes, when there is much wasting or when the urine gives a marked reaction with perchloride of iron, there is great danger of coma developing. In such cases it is important to remember that a long railway journey, over-exertion, or sudden change of diet are liable to be followed by coma. Prolonged constipation is also dangerous. As already mentioned, the diet ought not to be too rigid. Some carbohydrate food (especially bread) ought to be allowed. Cream and fatty food should be given freely. Whenever there are signs of commencing coma the carbohydrates in the diet should be increased a little and the nitrogenous food diminished. Constipation should be relieved by the use of purgatives, and Schmitz believes that he has checked the onset of coma by producing free action of the bowels with castor oil.

In severe cases, especially when the perchloride of iron reaction is intense, alkalies should be given in large quantities—400 to 500 grains of sodium bicarbonate in the twenty-four hours. Naunyn believes that this treatment is of great importance, and that the onset of coma may sometimes be prevented thereby for a long period. When early symptoms of coma are observed, alkaline treatment ought to be commenced at once, if it has not been prescribed previously. A number of cases are now on record in which the early symptoms of coma have subsided under vigorous alkaline treatment.

Sodium bicarbonate may be given in a little milk or in an effervescing draught with a little citric acid and saccharine (Yeo); or citrate of sodium may be given (900 grains in the twenty-four hours, Lépine).

During the last ten years the intravenous or subcutaneous injections of warm alkaline or salt solutions, as recommended by Stadelmann, have often been tried when coma has become well marked. There can be no doubt (1) that this method of treatment has often a decidedly beneficial effect, but (2) that the results are usually only temporary, and a fatal termination is very rarely prevented.

Solutions which have been chiefly used are:—A 3 to 5 per cent solution of sodium bicarbonate in 0·6 to 0·7 per cent sodium chloride solution, a 3 per cent watery solution of sodium bicarbonate, a solution of 10 grammes of sodium bicarbonate and 7 grammes of sodium chloride in a litre of sterilised water (Lépine), a 0·6 per cent solution of sodium chloride (v. Noorden). Intravenous injection (into the median basilic vein) is to be preferred to subcutaneous injection. It is important that the fluid should be warm (Lépine recommends a temperature of 38° C. = 100·4° F.) A large quantity of fluid should be used (2½ pints Oliver, 2 litres Lépine).

Usually the only result is an improvement in the pulse and a diminution of the coldness of the skin and a little diminution of the coma, so that the patient can be more easily roused. But occasionally the coma disappears partially or completely, so that the patient can converse with his friends; usually a relapse soon occurs.

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Diaphoretics.—The term diaphoretics is applied to all measures which increase the secretion of sweat.

The Physiology of Sweating.—The activity of the sweat glands is closely related to the condition of the cutaneous circulation, and therefore to the heat-regulating mechanism. When the surrounding atmosphere is warm the cutaneous vessels dilate, the amount of sweat is increased, and heat is lost by the resulting evaporation. When the atmosphere is cold the vessels of the skin contract and the amount of sweat is decreased. The sweat glands are not, however, actually dependent on the cutaneous circulation, but their activity is under the influence of a nervous mechanism analogous to that presiding over other secreting organs. This fact is supported not only by experimental evidence, but by such evidence as the occurrence of sweating from mental emotion, the sweating brought about by a venous condition of the blood, or the reflex sweating which results from the introduction of pungent substances into the mouth.

Enumeration and Mode of Action. — The principal diaphoretics are warmth; hot drinks; jaborandi (pilocarpin); liquor ammoniæ acetatis or citratis; potassii citras; potassii nitras; ipecacuanha (Dover's powder); opium; antimonium (pulvis antimonialis or vinum antimoniale); alcohol; salicin and the salicylates; various pungent and aromatic substances.

We find the simplest means of producing diaphoresis in the direct application of heat to the skin, whether by means of warm baths, of hot air or vapour baths, or the various kinds of pack. The sweating appears to be brought about not by direct stimulation of the sudoriferous glands, but through the action of the central nervous system. It may be greatly aided by the administration of hot drinks, possibly owing to an increased flow of warm blood through the nerve centres; and these, further, may be stimulated reflexly by the addition to the drinks of pungent or spicy substances. Alcohol produces diaphoresis chiefly by dilating the cutaneous vessels and increasing the circulation through the skin, but it also stimulates the nervous mechanism. Like other narcotics, such as opium, it produces sweating in the later stages of its action by increasing the venosity of the blood. Pilocarpin produces copious sweating chiefly by stimulating the peripheral ends of the secretory nerves, while nicotine produces a similar result chiefly by acting on the central nervous system.

Uses.—Diaphoretic measures are largely used on account of their antipyretic effect in pyrexia associated with the onset of acute specific fevers, or the occurrence of acute local inflammations. For example, at the onset of an attack of acute nasal or bronchial catarrh the administration at bedtime of a small dose of opium combined with other diaphoretics will often be found to greatly relieve both the local and the general discomfort. A quarter of a grain of morphia may be ordered in a glass of hot toddy, or from 5 to 10 grains each of Dover's powder and phenacetin. This should be followed on the next day by the administration of such diaphoretics as liquor ammoniæ acetatis and spiritus ætheris nitrosi; antimony, ipecacuanha, and occasionally aconite are useful in similar cases.

In some skin diseases associated with deficient activity of the sweat glands, diaphoretics are of assistance in the treatment.

Perhaps the most important use of diaphoretics has arisen from the recognition of the fact that the secretory action of the skin may be to a certain extent vicarious. Hence the value of diaphoretic measures in acute or chronic nephritis with suppression of urine or indications of the supervention of uræmic symptoms. Here the use of the hot mustard pack or the hot vapour bath, with such adjuvant measures as have been already mentioned, may be sufficient, but in many cases the powerful aid of pilocarpin is called for and proves of the greatest value. It is best administered in small doses hypodermically, the patient meanwhile being warmly covered in bed. If there is any weakness of the heart, stimulants (sal volatile) should at the same time be administered internally.

Diaphragm.

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The Diaphragm, or Midriff, the partition between the thorax and the abdomen, is a double muscle, the two sides of which, though capable of independent contraction, act in health habitually together. Its muscular fibres arise from the lumbar vertebræ by means of the crura, from the arched ligaments, from the cartilages of the six lower ribs, and from the posterior surface of the ensiform cartilage. Arching upwards and inwards these fibres converge on the tendon in the centre. Over the lower surface of the diaphragm is spread the peritoneum; over its upper the pleuræ and pericardium. It is perforated in various places, and this allows of the passage through it of the aorta, the œsophagus, the vena cava, the splanchnics, the sympathetic, etc. The diaphragm is supplied mainly by the phrenic nerves (each nerve controlling one lateral half), and perhaps to some small extent by the lower intercostal nerves.

During inspiration the two lateral halves of the diaphragm contract simultaneously, thus diminishing the curve of the arch and increasing the capacity of the thorax. The type of breathing in women being chiefly costal, the diaphragm is not so much used by them, and its excursions are not so well marked as is the case in men.

Apart from the results of disease of the phrenic nerves, the height at which the diaphragm stands is dependent on the relation of the pressure in the thorax to that in the abdomen. When this relation is disturbed the diaphragm is pressed upwards or downwards according as the balance of pressure is greater on the abdominal or on the thoracic side. But as this displacement is of interest, not from the point of view of the diaphragm, but from that of the viscera which are affected, the subject will not be considered here. Nor shall we do more than mention that variety of pleurisy in which the pleural covering of the diaphragm is affected. The same remark applies to peritonitis, in which the peritoneal covering of the diaphragm is implicated. In both cases the muscular fibres may show some degree of inflammation.

No tumours originate primarily in the diaphragm, but that structure is often the seat of secondary growths. Tuberculous and malignant processes are apt to pass from the peritoneum through the diaphragm to the pleura, more usually, I think, on the right side of the body than on the left. The muscular fibre of the diaphragm is liable, like other striped muscle, to suffer from fatty infiltration and from fatty and hyaline degeneration, and most probably rheumatism may also attack it. Gastric ulcers, in which the peritoneal surface of the stomach having become inflamed, has attached itself firmly to the serous surface of the diaphragm, may make their way through that structure.

The main interest of the diaphragm from a medical point of view lies in the fact that it is an index to the state of the phrenic nerve and its centre. Under one set of conditions you may have paralysis, under another spasm.

Paralysis—Apart from that paralysis sometimes seen in hysteria, and which is usually of little moment, the muscle may be paralysed by any lesion of the phrenic nerve or of its centre. Whether the paralysis affects one or both sides of the diaphragm depends, of course, on whether one or both phrenic nerves are implicated. Traumatic lesions of the upper cervical vertebræ, tuberculous processes there, tumours, inflammatory or other swelling of the meninges, may so interfere with the phrenic nerves as to stop the conduction of motor impulses and cause paralysis of the diaphragm. In their long and deep course through the neck these nerves, though protected from ordinary injury, may be divided in cases of wounding, or seriously compressed by tumours. Even in their course through the thorax the phrenic nerves sometimes suffer from the pressure of mediastinal tumours or are involved in inflammatory processes.

But, more commonly, paralysis of the diaphragm is due to some affection of the whole neuron. Any of the poisons, known and unknown, which produce neuritis, may affect the phrenic nerve. Paralysis of the diaphragm is believed to arise from rheumatism, though this must be a rare occurrence. It is seen more commonly in diphtheria, in alcoholic neuritis, and in beri-beri, and the toxin of influenza may produce like effects. The phrenic nerves may be involved in cases of lead poisoning; and in progressive muscular atrophy, amyotrophic sclerosis, and bulbar paralysis, the motor cells of the phrenic nerves are sometimes affected and the diaphragm in this way paralysed. In cases in which the pleural or the peritoneal surfaces covering the diaphragm are inflamed, a certain degree of paralysis results. To some extent the defective movement of the diaphragm in these cases is no doubt voluntary, and owing to the pain which movement occasions, it is probably also in part reflex. But it may be due in some measure to the inflammatory process penetrating from the serous covering to the substance of the diaphragm, and there involving the small nerve branches in such a way as to cause paralysis.

Symptoms.—When both sides of the diaphragm are paralysed there is no longer to be observed the normal protrusion of the epigastrium on inspiration, indeed there is sometimes a sinking in in that region. During expiration, on the other hand, the epigastrium protrudes. When only one phrenic nerve is paralysed these signs are unilateral. So long as the patient lies perfectly still there is little or no dyspnœa, though the rate of breathing is somewhat accelerated. But on the least exertion dyspnœa at once shows itself.

Litten has recently pointed out that the normal movements of the diaphragm may be readily recognised, especially in men, if the thorax be carefully watched about the sixth intercostal space. As the diaphragm separates itself from the thoracic wall a shadow moves downwards in a wave-like fashion over two or three interspaces, and again returns during expiration. To recognise this phenomenon the patient should be placed in a recumbent posture with the feet towards a good light. The observer should stand about three feet off with his back to the light. This sign may be of great importance in investigating doubtful cases.

Save in hysterical cases diaphragmatic paralysis is always of grave import, because any pulmonary complication, even of a light nature, is thereby rendered dangerous.

The *treatment* is that of the malady in the course of which the diaphragmatic paralysis has appeared. Blisters or hot applications over the course of the phrenic nerve in the neck are said to do good. Occasionally stimulation of these nerves by means of the faradic or of the galvanic current may be beneficial.

Spasm of the diaphragm may take either the tonic or the clonic form. Of these the latter is the more common.

Tonic spasm of the diaphragm occurs in bronchial asthma. The centripetal impulse passing up the vagus to the respiratory centre is reflected down the phrenic nerve, and so causes a tonic contraction of the diaphragm, a condition clinically recognisable by lowering of the inferior borders of the lungs. Apart from that occurring in asthma, tonic spasm of the diaphragm is an exceedingly rare affection. It has been known to accompany rheumatism, and to occur in cases of tetanus and tetany. When the diaphragm passes into tonic contraction the symptoms are very striking. The lower thorax expands, the epigastrium protrudes, the liver is pressed down, and the lung border is lowered. Along with these signs there develops an extreme and dangerous dyspnœa. To counteract the descent of the diaphragm the abdominal muscles contract powerfully; and to diminish the dyspnœa the extraordinary muscles of respiration come into vigorous action. The patient also complains of severe pain round the thorax at the level of the diaphragmatic attachment.

The condition is a serious one, and may prove fatal. The treatment must, therefore, be energetic and rapidly carried out. Some relief may be obtained by means of fomentations and sinapisms, and in a hot bath the spasm may relax. Duchenne advised the use of the faradic brush over the skin in the neighbourhood of the diaphragm. The administration of the nitrites, of chloroform, or of a morphia injection may be rapidly beneficial.

Clonic contraction of the diaphragm, commonly known as hiccough, is much more common than tonic spasm. The contractions, which are usually fairly regular, vary much in rate, being sometimes as slow as four or five in the minute, and sometimes as frequent as one hundred. The spasm is not usually limited to the diaphragm. The glottis is also narrowed and the nares move. Such constantly recurring diaphragmatic contraction has of necessity a considerable effect on respiration. If the rate of the hiccough is rapid, there may be considerable dyspnœa, and even the act of swallowing may be interfered with. The patient is shaken by each contraction, there is considerable pain along the diaphragmatic attachment, and much exhaustion may result. If the attack be prolonged—and some last for weeks—the condition may prove very serious.

Hiccough is due to irritation of the respiratory centres, and this irritation may be peripheral or central. The *peripheral causes*, which are the commoner, include such sources of irritation in the alimentary tract as are produced by stricture of the œsophagus, by overloading of the stomach, by gastric or intestinal affections, by appendicitis, by peritonitis, and by affections of the liver. Disease of the bladder, of the uterus, or of the prostate may, in the same reflex way, induce hiccough. Irritation of the pleura or of the pericardium may act in a similar fashion. *Central causes* are seen in cases arising from cerebral or spinal lesions, and in those which follow strong mental emotions, such as fear or anger. Poisoning of the nerve centres is probably the method of origin of that hiccough which shows itself in such diseases as typhoid fever, septicæmia, and uræmia, and of the form which so frequently precedes the lethal exit. It is to be remembered that in most cases of hiccough aneurotic predisposition is present.

As to *treatment*, it is clear that efforts should be made to remove the cause, if that can be recognised. But, apart from this, it will often be found that other forms of excitation of the nerve centres may bring the attack to an end. Sneezing may do so, for example, or coughing, or the exhibition of an emetic. Holding breath after inspiration, and making a strong expiratory effort while the glottis is kept closed, may be of benefit. The writer has seen steady pressure over the phrenics do good in an obstinate case of hiccough. Galvanisation of these nerves should also be tried, the positive pole being applied over the nape of the neck, and the negative over the phrenic. Hot fomentations applied round the lower thorax and over the epigastrium sometimes do good, or counter-irritation may be tried over these areas with benefit. In serious cases it may be needful to resort to morphia, to atropine, to chloroform, or to nitrites.

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SURGICAL ANATOMY.—The diaphragm is a large dome-shaped, musculo-tendinous structure separating the thoracic and abdominal cavities. Its muscular elements take origin below from the bodies of the upper three or four lumbar vertebræ and their intervening discs, from the arched ligaments which extend thence to the transverse processes and tip of the last rib, from the cartilages of the lower six ribs, and from the back of the sternum. They converge to be inserted into the strong central tendon, which is the highest portion of the diaphragm, lying about the level of the lower edge of the sternum, or of the seventh chondro-sternal articulation. The vault of the diaphragm reaches to the level of the fifth rib on the right side, and not quite so high on the left. Its nervous supply is derived from the phrenic and sympathetic nerves.

On the thoracic side the pleura and pericardium overlie the diaphragm; while the peritoneum lines its abdominal aspect.

The continuity of structure of the diaphragm is interrupted by three large openings for the transmission of the aorta, the inferior vena cava, and the œsophagus, and by smaller foramina for the vena azygos minor, the splanchnic, and the sympathetic nerves. Any one of these openings may be unduly large and so predispose to hernia, that for the œsophagus being the most important in this connection. As bearing upon this point also, as well as on the question of perforation of the diaphragm by inflammatory products, Tillmann has drawn attention to the fact that at certain points the muscular tissue is often deficient, so that the pleura and peritoneum alone keep up the integrity of the arch. Although these gaps vary greatly in size, shape, and situation in different individuals, their presence is fairly constant, the most important being (1) that between the spinal and costal origins of the muscle, in relation to which are the kidney, with its surrounding connective tissue, and the liver; (2) that between the costal and sternal fibres, over which the pleura lies on the right side, and the pericardium on the left; (3) sometimes the sternal fibres are entirely awanting, so that a wide space is left, over which lies the anterior mediastinum.

In some cases the lower part of the diaphragm is exceedingly thin; more rarely one half, or even the whole of the muscle is awanting.

INJURIES.—Although traumatic lesions of the diaphragm are by no means uncommon, they seldom occur apart from injuries of the viscera which lie in its immediate vicinity, and it is the signs of damage to the thoracic or abdominal organs which dominate both the pathological and the clinical picture in these cases.

Open wounds are usually the result of gun-shot injuries, punctures, or sabre cuts. As the lung does not extend down as far as the pleura, or the pleura as far as the diaphragm, it is possible for the diaphragm to be wounded alone, or along with the pleura, while the lung escapes.

Subcutaneous rupture is commonest after falls or crushes, but may result from severe muscular efforts, such as those of vomiting or parturition. The jagged end of a fractured rib, or even an unskilfully manipulated œsophageal bougie, has been known to perforate the diaphragm.

As this muscle never is, and never can be, at rest, its wounds either fail to close, or heal with a thin, weak, and stretchable cicatrix, which readily favours the formation of a hernial protrusion.

Clinical Features.—So far as the symptoms of injury to the diaphragm itself can be dissociated from those of the concomitant visceral lesions, they would appear to be *localised pain*, which is aggravated on deep respiration or coughing, and which leads to the patient restraining the action of the muscle as far as possible. *Bleeding* may take place from open wounds either externally or into the thoracic or abdominal cavities. *Shock* may be so severe as to end fatally. *Risus sardonicus*, which is supposed to be peculiarly related to morbid conditions affecting the diaphragm, may be present.

As a matter of fact, however, the *diagnosis* of an uncomplicated injury to the diaphragm is exceedingly difficult, and in the case of subcutaneous rupture all but impossible.

The *treatment* can only be discussed along with that of the associated lesions of the lung, pleura, pericardium, stomach, etc. (*q.v.*) One of the great risks attending such injuries is the strangulation of any portion of the alimentary canal which may become herniated, either at the time of the accident or long afterwards. So real is this danger that Stephen Paget advocates a systematic attempt being made to close such ruptures by introducing sutures from the thoracic side of the rent. The *mortality* of wounds of the diaphragm is exceedingly high (29 out of 33 cases ending fatally according to Frey), peritonitis, empyæmia, shock, or hæmorrhage being the commonest causes of death.

DIAPHRAGMATIC HERNIA.—Any protrusion through the diaphragm is spoken of as a diaphragmatic hernia, although in the vast majority of cases —about 88 per cent—there is no hernial sac, and the condition is rather one of prolapse than of true hernia.

Morbid Anatomy.—The protrusion may take place (1) through one or other of the natural openings in the diaphragm, particularly that for the œsophagus; (2) through one of the congenital deficiencies in the muscle, described by Tillmann; (3) through a rupture produced by indirect violence or muscular effort; or (4) through a direct wound of the muscle.

Lacher, to whose researches we owe much of our knowledge of this subject, found that in 276 cases collected by him, only 28 presented a sac, and of these 25 were congenital; and in Bohn's collection of 80 congenital cases, only 14 had a sac. In round numbers, therefore, about 20 per cent of congenital cases have a sac, and are therefore true herniæ, while only about 2 per cent of traumatic cases can be so described.

That the great proportion of diaphragmatic protrusions, whether with or without a sac, should be situated on the left side is natural, when we consider the substantial support given to the opposite side by the liver, the fact that all the large anatomical openings, as well as the congenital deficiencies are towards the left side, and that most suicidal and homicidal wounds are aimed at the heart.

Of 150 traumatic cases, 127 were left-sided, and of 117 of congenital origin, 98 were on that side.

The greater liability of men to accidental and other forms of injury may explain the fact that diaphragmatic hernia is five times commoner in the male sex than in females.

The opening is usually far back through the tendinous part of the diaphragm, but may be met with elsewhere, particularly through the opening for the œsophagus. It varies in size and shape from a mere slit or rounded aperture to a complete absence of one half of the muscle.

As a rule, portions of more than one viscus are prolapsed. The relative order of frequency may be gathered from the following:—Stomach, 151 times; colon, 145 times; small intestine, 83 times; liver, 45 times; duodenum, 35 times; pancreas, 27 times; cæcum, 20 times; kidney, 2 times.

It is interesting to observe, from the point of view of operation, that adhesions between the different prolapsed viscera or to the diaphragm seldom occur.

Clinical Features.—Children born with a *congenital* diaphragmatic hernia seldom live long enough to manifest symptoms which lead to a diagnosis. If not still-born, they usually survive but a few hours or days, and the abnormality is only discovered on post-mortem examination.

On the other hand, it is not uncommon for a large diaphragmatic hernia to be found at the autopsy on a person who has never manifested any of the ordinary clinical features of such a condition. In all cases, moreover, the diagnosis is one of extreme difficulty, and appears to have been made with accuracy only in some seven out of nearly three hundred cases.

The prominent features in well-marked *cases of some standing* are (1) an unnatural depression in the upper part of the abdomen, with a corresponding fulness in the lower thoracic region. (2) Well-marked signs of air in the pleural cavity, simulating those of pneumo-thorax, are present, varying in extent and degree with the amount of the alimentary tract which has been displaced, and the particular viscera involved. Leichtenstern has suggested that the amount of stomach in the hernia may be estimated by filling it with water or air from the mouth, or the colon from the rectum. Distinct intestinal gurgling may sometimes be heard on auscultation of the chest. (3) The heart may be displaced and its action interfered with, causing palpitation, attacks of dyspnœa, and a feeling of oppression in the chest, with inability to lie on the affected side and cough. These phenomena also vary in intensity with the size and nature of the herniated viscera. (4) Vague and atypical symptoms of dyspepsia are prominent features of all these cases. Pain in the epigastrium, heartburn, nausea, vomiting, thirst, or constipation alternating with diarrhœa are commonly complained of. Sometimes these symptoms are worst after exertion;

occasionally a full meal gives temporary relief; and in some instances the patient is conscious that the food lodges in the region of the chest, where it produces a fixed pain.

When the hernia is *suddenly developed* the predominant clinical feature is intense dyspnœa with severe precordial pain, a sense of oppression, and an inability to lie on the affected side, irritative cough, and a sensation that something has given way. Shock is marked, and may prove fatal, or death may result from compression of the lung.

A diaphragmatic hernia is liable to become *strangulated* either at the time of its production, or at any time thereafter. Some violent muscular effort usually determines this event, but in some cases it has been inexplainable. The patient presents all the clinical features of acute intestinal obstruction, without, as a rule, any guiding symptom to indicate the seat of strangulation. Hence the condition is seldom recognised during life. Rupture of gangrenous bowel into the pleural cavity will give rise to an empyæma, and a cure may follow its evacuation by thoracotomy.

Treatment.—An established hernia without symptoms of strangulation is so seldom diagnosed that the question of deliberate surgical intervention scarcely arises. It is only in cases of recent wounds of the diaphragm, with protrusion of viscera, and in cases of strangulation, that treatment is possible, and then it is only by operation that any good can be done.

(*a*) In *recent traumatic cases* an attempt should always be made to restore the displaced viscera, after thorough purification, to their place in the peritoneal cavity. For this purpose the wound may be enlarged as far as necessary, and ribs resected sufficient to give free access. The rent in the diaphragm should at the same time be closed, and the opinion of authorities is unanimous that this can best be done from the thoracic side.

When symptoms of strangulation are present, other points arise for consideration. The principles which guide the surgeon under these circumstances are discussed in the next paragraph, *a propos* of strangulation of an established diaphragmatic hernia.

(*b*) Strangulation of an established diaphragmatic hernia is so seldom diagnosed that in nearly every case any operation which is performed will be in the form of an exploratory laparotomy. Even then it is by no means certain that the seat of constriction of the bowel will be detected, and numerous cases are on record where this has only been found after death. When the operator is fortunate enough to recognise the obstruction, however, authorities are agreed that the pleural cavity should at once be opened, by a U-, T-, or H-shaped incision, with resection of ribs, and the herniated bowel exposed before any attempt at reduction is made, for the following reasons. (1) In this way the fatal error of drawing a piece of ruptured or gangrenous bowel into the peritoneal cavity in a situation so inaccessible as the vault of the diaphragm will be avoided. (2) The condition of the prolapsed viscus can be determined, and steps taken to purify or repair it, as may be necessary, before its replacement. (3) It is found to be both easier and safer to reduce the hernia by pushing from above than by pulling from below. (4) The facility with which reduction is effected from above is doubtless due to the fact that the admission of air abolishes the negative pressure in the pleural cavity. (5) The thoracic opening permits of the purification and drainage of a soiled pleural cavity, and so diminishes the risks of empyæma. (6) The closing of the opening in the diaphragm is only possible if attempted from the upper aspect.

While the opening of the pleura has these surgical advantages, it must at the same time be remembered that it adds to the already serious condition of the patient the risks incident to pneumo-thorax, and may turn the balance against him.

INFLAMMATORY AFFECTIONS.—*Diaphragmatic pleurisy* often closely simulates the onset of acute perforative peritonitis and other surgical abdominal conditions.

An *abscess* may form in the substance of the diaphragm, as occurred in Meltzer's case, where a young child suffering from pneumonia developed symptoms simulating those of empyæma. After exploratory puncture with negative result, the chest was opened, and a localised abscess found in the diaphragm.

Perforations, however, are much more common, the diaphragm becoming secondarily involved in inflammatory processes originating in neighbouring organs.

The primary seat of disease may be in the thorax, in the form of empyæma, abscess, or gangrene of the lung, tuberculosis, suppurative pericarditis, or mediastinitis. After breaking through the diaphragm the inflammatory products may enter the peritoneal cavity, giving rise to a sub-phrenic abscess; or may pass between the peritoneum and the muscles of the back as a lumbar abscess, eventually opening to the skin. Where adhesions have formed between the diaphragm and some portion of the alimentary canal, a purulent collection, such as empyæma, may discharge itself into the bowel, and be voided by the rectum.

On the other hand, the diaphragm may be perforated from below, a sub-phrenic abscess thus finding its way into the pleural cavity, lung, mediastinum, thoracic wall, pericardium, or even into the heart itself. The perforation usually takes place through one of the abnormally thin portions of the muscle, through one or other of the anatomical openings in the diaphragm, or at the seat of adhesions.

SUB-PHRENIC ABSCESS may originate in such a variety of conditions that it is by no means a rare affection. Owing to the frequency with which such collections contain air the condition is sometimes referred to as a "sub-phrenic pyo-pneumo-thorax," a nomenclature which involves a contradiction in terms. Maydl's table indicates the different primary causes of sub-phrenic abscess, their relative frequency, and the proportion of cases in which there is air in the abscess cavity.

Out of 179 cases collected by him, sub-phrenic abscess originated—

In the stomach and duodenum	in 35,	of which	20	contained air.
,, ,, cæcum and appendix	,, 25,	,,	8	,,
,, ,, liver or biliary passages	,, 20,	,,	1	,,
,, internal injuries	,, 18,	,,	3	,,
,, hydatid disease	,, 17,	,,	3	,,
,, the intestines	,, 13,	,,	4	,,
As a metastasis	,, 11,	,,	1	,,
In inflammation round the kidney	,, 11,	,,	1	,,
,, miscellaneous conditions	,, 11,	,,	5	,,
,, disease inside the chest	,, 9,	,,	1	,,
,, external injuries	,, 6,	,,	0	,,
,, caries of ribs	,, 3,	,,	0	,,

These collections may be situated anywhere between the liver and the diaphragm, but are commonest in the left hypochondrium.

Many of these primary conditions may perforate the diaphragm without first forming a sub-phrenic abscess. Thus Pick found that out of 28 cases of gastric ulcer leading to perforation of the diaphragm, 20 did so directly, while only 8 formed a sub-phrenic abscess. Ulcers of the fundus of the stomach, especially if associated with adhesions, tend to end thus more than others. Ulcers on the posterior part of the stomach by contracting adhesions obliterate the lesser sac of the peritoneum, so that when perforation takes place a sub-phrenic abscess results.

In appendicitis the pus may reach the diaphragm on the inner side of, or behind, the peritoneum. The sub-phrenic abscess may develop very rapidly in acute suppurative cases, with perforation or gangrene of the appendix, especially when no adhesions have formed around the cæcum; but as a rule it does not occur for some weeks. Unless such an abscess is evacuated, early perforation of the diaphragm is almost sure to follow. Thus out of 25 cases 11 were not operated upon, and all perforated. Of the 14 which were incised 9 were saved.

Clinical Features.—The comparative frequence with which air occurs along with the pus renders sub-phrenic abscess very liable to be mistaken for pneumo-thorax or pyo-pneumo-thorax; while many of the cases in which there is no air closely simulate empyæma.

The onset may be acute, especially when due to perforation of a gastric ulcer, or there may be little or no evidence of the formation of a very large abscess.

In addition to the general signs of pus formation, rapid or slow as the case may be, there is usually an excessive fulness and resistance in the region of the epigastrum or left hypochondrium, which often tends to point by the side of the ensiform cartilage.

The liver dulness may be lost on account of the gas in the abscess cavity, and the liver is often markedly displaced downwards by the pus. The diaphragm, too, may be pushed up as far as the third or even the second rib. The heart also is often displaced.

Clinical evidence of the existence of one or other of the conditions which give rise to sub-phrenic abscess, such as gastric ulcer, hepatic abscess, hydatid disease, empyæma, etc., is of great diagnostic value.

As distinguishing this condition from true pyo-pneumo-thorax, it is found that the hyper-resonant note is lower in the thorax, and extends into the upper abdominal regions, the pus lying still lower. Amphoric breathing and the bell-sound may be present.

When a sub-phrenic abscess has perforated into the pleural cavity, the suddenness of the onset of thoracic symptoms, with a previously healthy condition of the chest, and the history of gastric, intestinal, liver, or other abdominal disease, render the diagnosis fairly clear.

The *prognosis* is on the whole unfavourable. If left alone it usually proves fatal by setting up empyæma, abscess of the lung, or peritonitis. Even when it opens into the alimentary canal a fatal issue is common.

Treatment.—The only rational and efficient treatment is free incision and drainage.

The causes, size, and site of these abscesses are so varied that no uniform method of operating is applicable to all. The physical signs, supplemented, if possible, by an exploratory puncture, will guide us to the correct situation for incision, which in all cases should be free, and should permit of thoroughly efficient drainage being established. The adhesions which form round the abscess usually shut off the general peritoneal cavity from infection.

When the pus is on the convex surface of the liver, or on the upper aspect of the kidney or spleen, access may be got through the pleura, after resection of portions of two or more ribs, with the overlying muscle. The diaphragm is divided, after the pleural sac has been shut off, by a circular series of stitches. It has been urged against this method that the pressure of the large tube necessary for drainage may cause necrosis of the adjacent ribs.

Although the results, even after incision, are not uniformly successful, it has usually been found that failure has been due either to co-existent complications, such as peritonitis, pneumonia, or metastatic purulent collections; or to incomplete operation, such as neglect to drain a concurrent empyæma, an outlying loculus of the abscess, or to inefficient drainage.

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Diarrhœa.

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DEFINITION.—The term diarrhœa, which means literally "a running through," is applied to the frequent discharge of loose evacuations from the bowels. The too frequent passage of motions of normal consistence is not properly spoken of as diarrhœa. Diarrhœa may be due to increased peristalsis, or increased intestinal secretion, or both. Although diarrhœa is, strictly speaking, a symptom only, there are many conditions where it is practically of sole importance, in which therefore it may legitimately be regarded as the disease.

ETIOLOGY.—Amongst etiological factors common to different forms of diarrhœa *age* is of considerable importance, for although diarrhœa may occur at any age, it is most common and much more fatal at the extremes of life. Children under the age of two years are very liable to diarrhœa (to the extent of fully 80 per cent of fatal cases), and in old persons a severe diarrhœa often occurs as a terminal complication in chronic wasting diseases. The *temperature* of the atmosphere is also of importance. A sudden marked fall in the temperature is apt to be attended by a number of cases of diarrhœa; while during the hot months of the year children suffer severely, and the mortality from diarrhœal diseases varies almost exactly with the mean temperature. While diarrhœa has not been shown to vary with the density of the population, *overcrowding, want of cleanliness*, and especially *contamination of the food* (*milk*) *supply* must be regarded as important etiological factors.

SYMPTOMS.—The passage of loose alvine evacuations with the accompanying discomfort may be the sole symptom present. More commonly gastric disturbance, pain, flatulence, and other symptoms are present as well.

Character of the Stools.—The *number* of the evacuations varies greatly. There may be only two or three in the day, the patient feeling quite well in the intervals; or there may be four, five, or more every hour, and the feeling of discomfort and desire for a passage may be constant. The *amount* of the evacuations also varies within wide limits, from a comparatively trifling discharge to so copious a flux as to rapidly drain the tissues of the body. The motions may be quite liquid and even watery, or may be of the consistence of thick gruel. They may contain scybalous masses resulting from preceding constipation. Fluid motions are often described as resembling "pea soup" (typhoid fever); "rice water" (cholera); "scrapings of meat" (dysentery); "frog-spawn" or "boiled sago" (dysentery with copious mucous discharge). In "lienteric" diarrhœa food taken shortly before is passed unchanged. The *colour* of the motions varies according to the amount of bile present, and also with the nature of the food. Commonly they are light or dark brown or clay-coloured. They may be black or tarry from the presence of blood, or may be coloured by various drugs (bismuth, iron, hæmatoxylin, etc.). The green colour so common in the diarrhœas of children may be due to biliverdin or to chromogenic bacteria (the green bacillus of le Sage). In some cases the evacuations have an extraordinarily offensive *odour* due to proteid decomposition. A cadaveric odour may develop in association with necrosis of mucous membrane. *Mucus* may be present in considerable abundance in catarrhal conditions of the large intestine, and the passage of mucous casts or "skins" is characteristic of a condition described as mucous disease or mucous colitis. *Fat* may be present in the stools in considerable abundance, sometimes largely in the form of fatty crystals, in association with deficiency of bile (so-called "acholic diarrhœa") or more characteristically of the pancreatic juice. Pus, gall-stones, enteroliths, intestinal parasites, fragments of gangrenous mucous membrane, or of malignant tumours, may be discovered in the motions (see "Fæces").

Pain.—Abdominal discomfort and even actual pain are present in most cases, usually most marked in the lower abdomen, and dependent on irregular peristalsis of the intestine and flatulence, parts of the intestine being spasmodically contracted, while neighbouring parts are over-distended by gas. The passage of the flatus is often attended by rumbling noises or borborygmi. Where there is much irritation in the rectum defecation is often attended by severe tenesmus.

Fever. — In ordinary simple diarrhœa fever is either absent or slight and transient, but many of the diseases which cause diarrhœa are highly febrile.

Constitutional.—When large liquid motions succeed each other rapidly as in choleraic diarrhœa the tissues are drained of fluid, the secretions, especially the saliva and the urine, are arrested, and the patient suffers severely from thirst, and soon sinks into a condition of collapse.

THE VARIETIES OF DIARRHŒA.—Diarrhœa is a symptom of so many different pathological conditions, that it is impossible to give any natural classification of the varieties met with. For convenience the following groups may be recognised:—

(1) A large number of cases are due to *local irritation*, arising especially from the ingestion of improper food. Here we may also include diarrhœa due to excess of bile, or to absence of bile; to intestinal concretions; to intestinal parasites; to purgative drugs, and to certain irritant poisons. A choleraic diarrhœa may result from arsenical poisoning. In these cases the irritation may lead to increased peristalsis, or to intestinal hyperæmia and catarrh, or to slight or severe inflammation of the mucosa.

(2) Certain forms of diarrhœa have long been recognised as *eliminative*, especially the diarrhœa of uræmia. Diarrhœa considered to be of this character has been described as occurring in rheumatism, diabetes, gout, and in certain fevers, especially confluent small-pox, influenza, puerperal fever, and streptococcic infections. Diarrhœa due to exposure to cold is possibly sometimes of this nature. Watery diarrhœa may be present during the disappearance of ascites or other effusions.

(3) *Nervous* (psychical) influences may give rise to diarrhœa, for example in persons about to undergo a surgical operation.

(4) Many instances of diarrhœa are *microbic* in origin. Under this heading are included those due to toxic substances arising from putrefactive processes in foods and beverages (meat poisoning, cheese poisoning, etc. The specific organisms of typhoid fever, cholera, cholera nostras, certain

forms of dysentery, tuberculosis may be found in the stools. Several outbreaks of diarrhœa have been attributed to forms of the bacillus enteritidis (Gardner). The diagnosis of malarial diarrhœa has been made by the discovery of Laveran's plasmodium in red blood-corpuscles contained in the stools. Diarrhœa has been attributed, perhaps somewhat doubtfully, to certain Rhizopoda (monadines) and Infusoria (Cercomonas intestinalis, Trichomonas intestinalis, Paramœcium coli).

(5) Diarrhœa may be *secondary* and associated with inflammation or even ulceration of the mucosa of the small or large intestine. Such inflammation may be (*a*) catarrhal, arising in the course of the specific fevers, as a terminal process in wasting diseases, or as a result of portal obstruction; (*b*) croupous or diphtheritic; or (*c*) ulcerative, as in tubercle, cancer, dysentery, typhoid fever.

Chronic Diarrhœa.—Chronic diarrhœa may follow an acute attack or may develop independently. It is usually associated with intestinal catarrh due to one of the conditions already mentioned. Intestinal catarrh associated with portal obstruction is not uncommon in elderly people, and is associated with diarrhœa, debility, and anæmia. Marked depression of spirits may also be present.

Mucous diarrhœa (mucous colitis) is a very chronic affection, but the severity of the symptoms varies greatly from time to time. It is characterised by the passage of the mucous casts spoken of above, and in some instances by severe enteralgia.

Tropical diarrhœa is a generic term applied to several forms of chronic diarrhœa which are apt to afflict Europeans who have been long resident in tropical climates.

DIAGNOSIS.—Occasionally patients complain of diarrhœa who do not suffer from diarrhœa at all, but simply from some local irritation about the anus or rectum. When diarrhœa is present the important point in diagnosis is to ascertain its cause. If the attack is acute it may be possible to trace it to some indiscretion in diet, or to some special article of food which may have given rise to intestinal disturbance in all who partook of it, or to exposure to cold. If the diarrhœa is very severe, and attended by pain and vomiting, the possibility of its being due to irritant poisoning, whether from putrefactive alkaloids or from inorganic poison, must be kept in view. Amongst articles of diet which have in many instances given rise to severe diarrhœa may be mentioned pork-pie, veal-pie, sausages, tinned meats and fruits, stale oysters, crabs, cheese, ice-cream.

In chronic diarrhœa a systematic examination of the evacuations, paying attention to the points mentioned in the paragraph on the "Character of the Stools," is of the first importance. Microscopic or bacteriological examination may lead to a correct diagnosis. Obviously the existence of any present or past disease which may be the cause of the diarrhœa must be ascertained. Digital examination of the rectum and examination with Kelly's rectal speculum will often give important information.

The diagnosis of the part of the bowel chiefly affected is usually difficult, and often impossible. In catarrh of the small intestine there are often severe colicky pains, and the stools are grayish-yellow or ochreous, not very frequent, and may contain partially digested food. There are no definite symptoms of duodenitis, but it is usually associated with gastritis, and often with jaundice. When the large intestine is affected, pain may be absent, or may be very intense. The evacuations have a soupy consistence, and may contain large quantities of mucus. Their passage may be attended by marked tenesmus if the lower part of the bowel is affected.

Treatment.—*Acute Diarrhœa.*—Acute dietetic diarrhœa requires little treatment in slight cases except rest and abstention from solid food, but in most cases recovery will be hastened by the administration of a purgative as soon as possible after the onset of the attack. For this purpose half an ounce of castor oil with 20 or 30 drops of laudanum is a favourite prescription. A dose of rhubarb is preferred by some. If the diarrhœa still continues twenty-four hours after the action of the purgative, an alkaline stomachic mixture with carminatives may be ordered, or moderate doses of aromatic chalk powder, chalk mixture, or bismuth. If vomiting is present as well as diarrhœa, ice may be given to suck, and small doses of bismuth and pulv. ipecac. co. administered at intervals of a few hours will be found useful. Where small motions are being passed frequently, and accompanied by a good deal of irritation of the rectum, an enema of one or two ounces of starch with 20 to 30 drops of laudanum should be given.

Other forms of acute diarrhœa should be treated on similar lines. Rest in bed, warmth, and the restriction of the diet to milk or bland farinaceous food often make the patient comparatively comfortable even when the evacuations have previously been very numerous.

In nervous diarrhœa, which is usually recurrent, the patient should be taught to restrain the impulse as much as possible. If about to be exposed to any condition likely to bring on an attack, a dose of bromide of potassium may be administered beforehand.

Chronic and Secondary Diarrhœa.—In all cases the diet must be attended to, and all articles of food which are not being properly digested must be forbidden. In some forms of tropical diarrhœa rest in bed and the absolute restriction of the diet to milk is found to be the most effective treatment. Prolonged rest in bed is often the best remedy for chronic diarrhœa in hysterical women.

A diet restricted more or less strictly to raw meat is found useful in some forms of chronic diarrhœa.

Where diarrhœa is associated with acidity of the intestinal contents, chalk or carbonate of bismuth may be ordered with some of the vegetable astringents, and with or without opium. In some cases, and especially when there are frequent watery discharges, the mineral astringents will succeed better, such as the pernitrate or perchloride of iron, sulphate of copper, or nitrate of silver.

Antiseptics have been much used of late in the treatment of diarrhœa of microbic origin, and may be tried in any case where the odour of the evacuations is very offensive. Perhaps the most successful method of producing some approach to asepsis in the bowel is to give one or two purgative doses of calomel followed by the continued administration of salol in full doses. Such treatment, however, is obviously unsuitable for many cases of chronic diarrhœa, but calomel in repeated small doses, carbolic acid, biniodide of mercury, naphthaline, β-naphthol, salicylate and subgallate of bismuth are all useful.

Where the mischief is chiefly in the colon, daily irrigation with a large quantity of fluid should be carried out. For this purpose salt solution (one drachm to a pint) or a 1 per cent solution of boracic or salicylic acid may be used.

If ulceration of the colon is present, astringent injections are often of the greatest value, such as nitrate of silver in the proportion of a drachm to two pints of warm water.

Literature.—*Articles:* "Choleraic Diarrhœa," "Colon," "Fæces," and "Gastro-Intestinal Disorders of Children."

Diathesis.—This term, derived from the Greek words *δια* and *τιθημί*, may be defined as the predisposition or constitution of the body, in virtue of which certain of its tissues or organs become, at one time or successively, the seat of affections similar in their nature, these affections presenting for their origin no other more potent or more definite cause. The nature and meaning of diathesis can probably best be explained by the following considerations.

In order that a human being should live through the normal number of years, doing during this period the ordinary amount of work, and exposed during this period to the ordinary risks from cold or injury, it is necessary that each organ and tissue in that being should possess, from the beginning, a proper amount of what is called vitality. It is evident that if any one of these tissues or organs has been endowed at the beginning of life with less than its proper amount of this vitality, its store must become exhausted previous to that of the others. A more or less localised breakdown will then occur, as the result of which the whole being will suffer, or perhaps even the continuance of life will be rendered impossible. Just as the speed of a fleet is dependent mainly on the speed of the slowest vessel in it, so the duration of life is dependent mainly on the store of vitality possessed by the weakest of the tissues or organs of the body.

It is known that, as the result of differences in the surroundings and modes of life amongst individual human beings, the wear and tear of life is not always equally distributed over the different tissues and organs. In one individual, for example, the heart and blood-vessels are specially strained, in another the bronchi and lungs, in a third the kidneys, in a fourth the nervous system, and so on. It is known, further, that the amount of vitality with which the new being is endowed at conception is dependent on the vitality of the parents. Hence it follows that, although the offspring may be always potentially better than the parents, if the parents have lived under unhealthy conditions, if they have been weakened by disease or accident, or if they have been immature or too old, then the store of vitality transmitted to all or certain of the tissues or organs of the offspring will be deficient. Hence it will follow that as the result of no special overstrain or no excessive exposure to cold or other morbid cause, a premature breakdown will occur. This, for obvious reasons, will tend to show itself specially in one or other of the tissues or organs, heart, lungs, nervous system, kidney, etc., and so, as the result of disease affecting one or other of those parts, the individual will be cut off long before the normal period of life is reached.

The precise nature of the disease changes which affects these parts is apt to vary. It may be an inflammatory change pure and simple, like a nephritis; it may be an inflammatory change due to the entrance of some organism, as a tuberculous phthisis or an endocarditis; it may be a so-called degeneration, as a fatty heart or an atheroma, or it may be a new growth, a cancer. In all cases, however, the great importance of the tissue vitality has to be borne in mind, and although it is always difficult, and ofttimes impossible, to say whether the surroundings or the constitutional condition has been the more important factor in causing a disease, there is no doubt that the capability of the physician to prognose and alleviate depends largely on his power of recognition and proper perception of the relationship between these two.

The duration of life being dependent on the power possessed by the tissues and organs of the body to maintain themselves against disease changes, and the diathesis being the predisposition to disease changes inherent in certain of these tissues or organs, it is evident that the number of diatheses should theoretically be very great. This number, indeed, should correspond not only with that of the tissues or organs, but also, at least to some extent, with the diseased conditions to which these tissues or organs are liable. Hence, as can readily be understood, a very great number of diatheses has been described. Among these the more prominent are the gouty, lithæmic, rheumatic, strumous, scrofulous or tubercular, cancerous, nervous, inflammatory, gangrenous, scorbutic, hæmorrhagic.

But a moment's consideration will show that this number is by far too large. Many of these are simply modifications of the same diathesis, others are to be regarded as diseased states rather than constitutional predispositions or diatheses. In this article, therefore, detailed reference need only be made to a few, and by this selection an additional advantage is obtained in that only those diatheses are described which are more or less readily recognisable by objective appearances.

Gouty.—In this diathesis the individual presents, at least until the middle periods of life, all the indications of robust vitality and of great bodily and mental activity. The frame is well built and well nourished, though tending perhaps somewhat to fatness. As might be expected, the digestive and assimilative powers are excellent, the teeth are good, the skin of the face is florid, over the trunk it is rather thick with active glandular organs, the hair is abundant, showing, however, rather early a tendency to turn gray. Owing to the excellent assimilative and digestive power, and the feeling of well-being and fitness which such a diathesis confers, gouty individuals are rather liable to excess in eating and drinking, and a tendency to a more or less plethoric condition supervenes, which in time gives rise not only to the special gouty changes in the fibrous tissues, but to the occurrence of allied morbid changes in the heart, arteries, notably the cerebral ones, urinary organs, etc. There is no doubt, however, that in its slighter degrees, should the tendency to excess be guarded against, this diathesis is one which is probably more than any other associated with long life and bodily and mental vigour. Insurance statistics seem to show that a family history of gout is often found among the best lives, and the saying of Sydenham that "more wise men than fools have suffered from gout" seems, if properly interpreted, to bear this out. A somewhat gouty habit is therefore not infrequently an advantage in life. Its disadvantages are indirect, being the result of its tendency to lead to excess, and consequently overstrain of the excretory organs.

Rheumatic.—This diathesis is one which to some extent resembles the gouty. The individual is usually well built, well nourished, and muscular, having good digestive and assimilative powers. But the blood and circulatory organs seem to present a lack of nutritive vigour. The cheeks are often ruddy, but it is to be remembered that this may mean the reverse of the exuberant health which it might at first sight indicate. Professor Laycock used always to teach his students to be distrustful of a very healthy-looking complexion.

In rheumatic individuals the innate weakness in the blood often shows itself in early life by the supervention of anæmia, especially in females, and the corresponding faulty condition of nutrition in the heart and blood-vessels by endocarditic changes. Moreover, as the result of comparatively trifling exposures, the characteristic inflammations of joints, serous surfaces, etc., are apt to arise.

Scrofulous.—This diathesis, which comprises the strumous and the tubercular, is one in which probably all the tissues and organs of the body have been endowed with an abnormally low degree of vitality, but in which, owing to this cause, the absorbent surfaces and the glandular organs are specially prone to suffer from the entrance into them of the tubercle organism. Its objective characteristics can probably best be described by following the older writers, and considering it as divided into two classes—(*a*) scrofulous, and (*b*) tuberculous (Jenner), or as these have been aptly enough, though not quite correctly, termed, the ugly scrofula and the pretty scrofula.

(*a*) *Scrofulous.*—Of this the following were the characteristics as given by the older writers—figure heavy, abdomen large, ends of the long bones rather large, shafts thick, skin thick and opaque, complexion dull and pasty-looking, face plain, lips and alæ of nose thick, lymphatic glands very perceptible to touch, temperament phlegmatic, mind and body lethargic.

Although certain distinct diseased conditions have been confused with the diathesis in this description, probably rickets and possibly cretinism, the above yet forms a picture which is, to a considerable extent, true to life.

(*b*) *Tuberculous.*—To this in its turn were ascribed the following characteristics. Figure slim, adipose tissue small in amount, ends of long bones small, shafts thin and rigid, limbs straight, skin thin and transparent, complexion clear, superficial veins distinct, blushing frequent, eyes bright, pupils large, eyelashes long, hair silken, face oval, good-looking. Children the subject of this diathesis are precocious, cut their teeth, run about, and talk early. In adults the nervous system is usually highly developed, and the mind and body are specially active, so that this diathesis is practically more or less identical with the nervous. It is not to be forgotten, further, that with it there is often associated a fair amount of muscularity and a considerable amount of physical strength. With it, as with the scrofulous, however, the constitution is faulty and the liability to the supervention of tuberculous diseases of all kinds is great.

Of the other diatheses the so-called cancerous, the scorbutic, and the hæmorrhagic require mention only. The first of these cannot readily be distinguished diathetically (see "Cachexia"); the other two are in reality forms of disease.

Diet.

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A FOOD may be defined as anything which, when taken into the body, is able either (1) to build up or repair tissues, or (2) to supply material for the production of heat or muscular work.

A true food must therefore be either a tissue-builder or a source of potential energy. Dietetic substances which are unable to fulfil either of these functions may have important actions on the body, but cannot be regarded as foods in the true sense of the term. Such substances are tea, coffee, and the extractives of meat.

THE NUTRITIVE CONSTITUENTS of foods may be arranged thus:—

Organic	Nitrogenous	Proteids, *e.g.* myosin of meat, casein of milk Albuminoids, *e.g.* gelatine
	Non-Nitrogenous	Carbohydrates, *e.g.* sugars and starch Fats, *e.g.* butter
Inorganic	Water Mineral matters, *e.g.* sodium, potassium; calcium and magnesium; iron; phosphorus, chlorine, sulphur	

The functions of food as building material, fuel, and a supplier of energy, are fulfilled by these different groups of nutritive constituents in varying measure.

Building material is supplied by the proteids, water, and mineral matters, and by these alone. Fats, carbohydrates, and albuminoids are unable to form tissue. Seeing that there is always a certain amount of waste of bodily structure going on there is a constant demand for these materials in the diet. Without them, or any one of them, life is impossible.

All the *organic* nutritive constituents of food serve as sources of heat. They are able to do so in virtue of their capability of undergoing oxidation in the tissues and organs. Fat requires the greatest amount of oxidation, and is therefore the most potent fuel food. Proteids, carbohydrates, and albuminoids are about equal as heat producers, but are scarcely of half as much value as fats. The standard of heat production is the Calorie, which means the amount of heat required to raise the temperature of one kilo. of water 1° Cent.

It has been found by experiment that—

One gramme of	proteid	when fully oxidised produces	4·1 Cals.
,, ,,	carbohydrate	,, ,, ,,	4·1 ,,
,, ,,	fat	,, ,, ,,	9·3 ,,

It is no doubt owing to their great value as heat producers that fats find such a large place in the dietary of the inhabitants of very cold climates, *e.g.* the Esquimaux. The presence of 40 per cent of fat in the milk of the walrus is an instance of a similar adaptation on the part of Nature.

As raw material for the production of *muscular energy* the carbohydrates are now admitted to rank first. It seems certain, however, that the proteids can either directly or indirectly serve a similar function, and probably the fats are able to do so too. Whether the albuminoids are able to fill a similar rôle is uncertain. The great utility of sugar especially, as a muscle food, has recently been insisted upon by many observers, and its value is confirmed from such diverse sources as laboratory experiments with the ergograph, the efficiency of troops on the march, and the experience of Alpine climbers. It will probably win for itself a larger place than it has as yet taken in the dietary of training. Notwithstanding this outcome of modern research the necessity of a liberal supply of proteid in the diet of those who have to perform severe muscular work must not be lost sight of. Strenuous muscular exertion is always accompanied, sooner or later, by an increased excretion of urea. Whether one regards this as arising from the utilisation of proteid as a source of energy, or ascribes it simply to the destruction of muscle fibre which severe work necessarily entails, the loss still requires to be made good. That is the physiological justification for the high opinion which trainers have always entertained of a richly proteid diet for those engaged in athletic contests.

It is evident that the proteids are, by themselves, capable of fulfilling all the demands upon a complete food. They are able to build and repair the tissues of the body, to supply it with heat, and provide the muscles with pabulum for their work. It is this physiological omnipotence which gives to the proteids that pre-eminence which they possess amongst the nutritive constituents of food, and thanks to it, life can be maintained on a diet of lean meat and water, as in the Salisbury cure, for a practically indefinite time.

THE AMOUNT OF FOOD REQUIRED IN HEALTH.—An idea of the quantity of food required daily in order to maintain the body in a state of health can be arrived at either by a physiological or an empirical method. The former is based upon estimations of the intake and output of the chief chemical constituents of the food, and of the amount of potential energy expended daily in the production of heat or muscular work. The latter is founded upon a simple analysis of the diets of healthy persons, living under known conditions, and neither increasing nor losing in weight. Dietaries based upon the results yielded by the first method are called *standard dietaries;* those which result from the second are *actual dietaries.*

(1) The chief chemical elements which the physiological method has to take into consideration are nitrogen and carbon. A healthy man, of average weight and doing a moderate amount of muscular work, excretes from 16-20 grammes of nitrogen and about 320 of carbon daily. In other words, he will require every day to be supplied with about ⅔ of an ounce of nitrogen, and ⅔ lb. of carbon, or one part of the former to every sixteen of the latter. The approximate proportions of carbon to nitrogen in some common articles of diet is as follows:—

Roast Beef	has	1 of Nitrogen to	5	of Carbon	Peas	have	1 of Nitrogen to	12	of Carbon
Eggs	have	,,	7	,,	Bread	has	,,	28	,,
Cheese	has	,,	10	,,	Potatoes	have	,,	33	,,
Milk	,,	,,	11	,,					

Evidently none has nitrogen and carbon in proper proportion. For this reason one must either adopt a mixed diet or consume an undue amount of one or other element. The former alternative is the one usually adopted, and is illustrated by the following standard English diet (Oliver):—

		Carbon.	Nitrogen.
Foundation—	1 lb. Bread . . .	117	5·5
	½ ,, Meat . . .	34	7·5
	¼ ,, Fat . . .	84	...
Accessories—	1 lb. Potatoes . .	45	1·3
	½ Pint Milk . . .	20	1·7
	2 Eggs	15	2
	2 ozs. Cheese . . .	20	3
		335	21

Such a diet more than supplies the amount of carbon and nitrogen required.

Taking the output of heat and work as the basis of calculation, it is found that a man doing moderate muscular work requires a supply of potential energy equal to 3000-3500 Calories daily. The next question is, what fraction of this total is to be provided in the form of proteid, carbohydrate and fat respectively? As regards proteid the reply is easy. The amount must be sufficient to provide 20 grammes of nitrogen. Now one part of nitrogen is contained in 6¼ of animal and 6-5·7 of vegetable proteid. Hence 20 × 6·25, *i.e.* 125 grammes of proteid will be an ample allowance. The relative proportions of carbohydrate and fat are not so easily determined, for we have no means of knowing how much of the total CO_2 excreted daily is derived from the oxidation of the one and how much from the other. All we can do is to distribute the balance of Calories not obtained from proteids between the two in accordance, as far as possible, with the results of actual observation of the usual amounts of each consumed by healthy men. As a matter of fact, the data given by different authorities on this point are conflicting, but 105 grammes of fat and 420 of carbohydrates may be regarded as a fair allowance, erring, if anything, in the direction of an undue preponderance of fat. These amounts will provide a total of 3211 Calories distributed as follows:—

			Calories.
Proteids	125 grammes	× 4·1 =	512·5
Fats	105 ,,	× 9·3 =	976·5
Carbohydrates	420 ,,	× 4·1 =	1722·0
			3211·0

Such a diet might be made up as follows (Atwater):—

	Oz.	Calories.		Oz.	Calories.
Beef	8	=560	Brought forward .		1945
Fish (mackerel or herring)	4	=230	Potatoes	8	=160
2 Eggs		=135	Rice	2	=205
Butter	2½	=565	Bread	9	=720
Cheese	1	=130	Sugar	1½	=175
Milk, 1 pint		=325			
			Total . . .		3205
Carry forward .		1945			

(2) A large number of *actual dietaries* have now been investigated, especially on the Continent and in America, the results of which are collected by Atwater in the following table:—

ACTUAL DIETARIES

Classes.	Nutrients.				Potential energy of nutrients.
	Proteid.	Fats.	Carbohydrates.	Total.	
European and Japanese dietaries.	Grms.	Grms.	Grms.	Grms.	Calories.
1. Sewing Girl, London, wages 93 cents. (3s. 9d.) per week	53	33	316	402	1820
2. Factory Girl, Leipsic, Germany, wages $1·21 per week	52	53	301	406	1940
3. Weaver, England, time of scarcity .	60	28	398	486	2138
4. Labourers, Lombardy, Italy; diet mostly vegetable	82	40	362	484	2192
5. Trappist monk, in cloister; very little exercise, vegetable diet . . .	68	11	469	548	2304
6. Students, Japan	97	16	438	551	2343
7. University professor, Munich, Germany; very little exercise . .	100	100	240	440	2324
8. Lawyer, Munich	80	125	222	427	2401
9. Physician, Munich	131	95	327	553	2762
10. Painter, Leipsic, Germany . .	87	69	366	522	2500
11. Cabinetmaker, Leipsic, Germany .	77	57	466	600	2757
12. "Fully-fed" tailors, England . .	131	39	525	695	3053
13. "Well-paid" mechanic, Munich, Germany	151	54	479	684	3085
14. Carpenter, Munich, Germany . .	131	68	494	693	3194
15. "Hard-worked" weaver, England .	151	43	622	816	3569
16. Blacksmith, England	176	71	667	914	4117
17. Miners at very severe work, Germany	133	113	634	880	4195
18. Brick-makers (Italians at contract work), Munich	167	117	675	959	4641
19. Brewery labourer, Munich; very severe work, exceptional diet . .	223	113	909	1245	5692
20. German soldiers, peace footing . .	114	39	480	633	2798
21. German soldiers, war footing . .	134	58	489	681	3093
22. German soldiers, Franco-German war; extraordinary ration . . .	157	285	331	773	4652

It will be observed that these correspond more or less closely, both in the proportions of their nutritive constituents and the total amount of potential energy yielded, with the physiological requirements already laid down and exemplified by the standard dietaries described above.

Roughly speaking, a man who adopts one of the above standard diets will consume about 3 lbs. of solid food daily, containing about 23 ozs. of dry solids, or nearly an ounce per hour. The amount of mineral matter and water which he requires will be considered later.

It must be clearly realised that such standard diets are only of limited applicability. They cannot be rigidly enforced in all cases and under all circumstances. They are useful, however, as furnishing one with some idea of the amount and kind of nutriment which should be supplied to a healthy man doing a moderate amount of work. Thus they are of great assistance in constructing diet tables for public institutions in which the inmates are unable to have a free choice of foods, as, for example, in prisons, workhouses, hospitals, and the like. In applying them to individuals respect must be had to various modifying factors, of which the chief are age and sex, height and build, work and rest, climate and surroundings, and, last but by no means least, personal peculiarity. The influence of each of these may now be briefly considered.

MODIFYING FACTORS.—(1) *Age and Sex.*—Children require relatively more building material, fuel and muscle food, than adults. They require more building material because they must not only keep their tissues in repair but add to them by growth; they require more fuel because, like all small animals, they have a large surface compared to their bulk, and so lose heat rapidly; they require more muscle food because of their restlessness and bodily activity in play.

Hence children require a large amount of food, and the importance of proteid and fat to them especially can hardly be over-stated. With the aged the reverse holds good. The assimilative power of their tissues is on the wane and their bodily activities restricted, hence their diet should be smaller than that of middle life. One may easily underfeed the young, but one is apt to forget that it is almost as easy to overfeed the old. Women require relatively less food than men, not only on account of the lighter nature of their work, but also, probably, from their possessing a slower rate of metabolism. Roughly speaking, a woman may be regarded as requiring only 8 parts of food to every 10 required by a man.

The proportions for children may be stated thus:—

A child	under 2	requires	0·3	the food of a man	at moderate work.
,,	from 3-5	,,	0·4	,,	,,
,,	,, 6-9	,,	0·5	,,	,,
,,	,, 10-13	,,	0·6	,,	,,
A girl	,, 14-16	,,	0·7	,,	,,
A boy	,, ,,	,,	0·8	,,	,,

(2) *Height and Build.*—These have a determining influence on the amount of food required by affecting (*a*) the body weight, (*b*) the extent of body surface. The greater the weight of the body the greater the amount of food required for its upkeep. Thus the heavy man requires more food than the light man.

The number of Calories which require to be supplied per kilo. of body weight are about as follows:—

During rest, 30-35.
„ light work, 35-40.
„ moderate work, 40-50.

The greater the extent of body surface the more rapidly heat is lost and the larger the supply of fuel required. For this reason the tall thin man who has a large surface requires more food than the short stout man who has, relatively to his weight, a much smaller surface.

(3) *Work and Rest.*—These two factors have a more potent influence on the amount of food required than any others. The necessity for a large supply of proteid and carbohydrate during the performance of severe bodily labour has already been pointed out. The number of Calories required under such conditions may rise to 4000 or even 4500 (for examples, see Table I.)

On the other hand, when the body is at complete rest, as is the case, for example, with an invalid lying in bed, the demand for potential energy may fall to 2000 Calories or less per day. Intellectual work does not appreciably increase bodily waste, and therefore does not demand a liberal diet. Quality, and especially easy digestibility, is a much more important consideration for the brain worker than mere quantity.

(4) *Climate* is probably of less real importance in determining the actual amount of food required than is commonly supposed. Fluctuations in external temperature should be met by diminishing heat loss through increase of the clothing rather than by increasing heat production by changes in the diet. Notwithstanding this, experience seems to show that in cold climates and in winter the fuel foods—and especially those rich in fat—should be increased, while in the reverse conditions the carbohydrates should be relatively increased, and the proteids and fats diminished. The blubber diet of the Esquimaux on the one hand, and the rice diet of the Hindu on the other, are examples in point.

(5) Personal peculiarities as affecting the amount of food required are largely to be explained by the influence of body weight and shape already considered. When all deductions are made, however, there is still room for the popular belief that some people can "get on" with less food than others, though both are living under the same conditions and performing the same work. Of this fact, if fact it be, there is no satisfactory explanation; but it is at least conceivable that some organisms are more economical machines than others, and turn their supplies of potential energy to better account, just as a man who has had practice will perform a given muscular feat with much less wear and tear than a novice. Such individual peculiarities may explain the tendency to obesity in certain families, and must always be borne in mind in regulating the diet of patients.

THE RELATIVE VALUE OF FOODS.—The relative value of different foods must be decided on chemical, physiological, and economic grounds.

(1) The chemical value of a food is determined by the results of its percentage analysis. The composition of most of the foods in common use is set out in detail in the tables in the later part of this article. It need only be remarked here that one cannot judge of a food from its chemical composition alone. A food which contains but a small percentage of nutrients can never be regarded as a valuable article of diet, but, on the other hand, the possession of high chemical qualities by no means necessarily implies suitability to the needs of the body.

(2) In judging of the value of foods on physiological grounds one has to consider (*a*) the digestibility of the food in the stomach, (*b*) its absorption in the intestine. A good food is one which is easily digested and well absorbed into the blood.

(*a*) The ease with which a food is digested in the stomach may be gauged by the time which elapses before it is passed on into the intestine.

This time is occupied in bringing the food into a state of solution and the more rapidly this is accomplished the greater is the digestibility of the food in question. As the results of experiments upon men the commoner foods may be arranged according to the length of time which they remain in the stomach as follows:—

1-2 hours.

7 oz. water or plain tea, coffee, or cocoa.
,, beer or light wine.
,, boiled milk.
,, beef tea.
Whites of 3 eggs.

2-3 hours.

¾ pint water, beer or boiled milk.
A large teacupful of coffee with cream, or cocoa and milk.
2 raw or poached eggs.
3½ oz. raw meat.
9 ,, boiled calves' brains or sweetbread.
2½ ,, raw oysters.
5 ,, boiled white fish.
7 ,, cauliflower or asparagus.
5 oz. boiled or mashed potatoes.
2½ ,, white bread, old or new.
2½ ,, rusks.
2 ,, biscuits.

3-4 hours.

8 oz. stewed chicken or roast partridge.
9 ,, boiled beef.
3½ ,, beef steak.
5½ ,, of any sort of bread or biscuits.
5½ ,, of rice, spinach, apples, or carrots.

4-5 hours.

9 oz. roast beef or steak.
9 ,, ,, goose.
10 ,, ,, duck.
5½ ,, mashed lentils or stewed French beans.
7 ,, ,, peas.

The articles in the first two groups are obviously those which make least demands upon the digestive powers of the stomach.

(*b*) The capability which a food has of absorption in the intestine must be clearly distinguished from its digestibility in the stomach. The most easily digested foods are by no means necessarily those which are most completely absorbed and *vice versa*. The following table (chiefly after Rubner) shows the degree to which the nutritive constituents of some typical foods are absorbed:—

Food.	Percentage Unabsorbed.			
	Dry Substance.	Proteid.	Fat.	Carbohydrate.
Rice	4·1	20·4	...	0·9
White Bread	4·2	21·8	...	1·1
Macaroni	4·3	17·1	...	1·2
Boiled Beef	4·9	2·6	...	...
Roast ,,	5·3	2·6	...	...
Eggs	5·2	2·6	4·4	...
Milk	8·8	7·1	5·3	...
Cheese	6·4	3·3	5·2	...
Maize	6·7	15·5	...	3·2
Peas	9·1	17·5	...	3·6
Whole Meal Bread	13·1	36·7	...	7·9
Potatoes (Mashed)	9·4	30·5	...	7·4
Cabbage	14·9	18·5	...	15·4
Carrots	20·7	39·0	...	18·2

It will be observed (1) that the carbohydrates and fats are much more completely absorbed than the proteids. Indeed, it may be said that the fat of the diet is absorbed almost entirely, while the only foods in which there is any appreciable loss of carbohydrates are green vegetables and, to a less extent, the pulses; (2) that vegetable proteids are not nearly so well absorbed as those of animal origin. It must not be supposed that it is desirable that a food should be completely absorbed into the blood. The contrary is the case. The intestine demands a certain amount of insoluble residue or "ballast" to serve as a stimulus to its peristalsis. Hence the advisability of selecting foods which leave a considerable residue for cases of constipation, and of those which are very completely absorbed in cases of diarrhœa. A study of the table will be of assistance in making such a selection.

(3) A good food must not only contain a high percentage of nutrients, and be easily digested and well absorbed; it must also be cheap. One may best arrive at the relative economic value of a food by calculating either the total number of Calories or the amount of building material obtained in it for a given sum. Thus one finds, proceeding on the first plan, that a shilling's worth of—

Wheat flour yields	13,782 Calories	Cheese yields	3,642 Calories
Oatmeal ,,	9,189 ,,	Butter ,,	3,082 ,,
Potatoes ,,	7,689 ,,	Milk ,,	2,178 ,,
Beans ,,	7,630 ,,	Leg of mutton yields	1,076 ,,
Fat salt pork ,,	6,825 ,,	Sirloin of beef ,,	870 ,,
Sugar ,,	6,292 ,,	Eggs ,,	768 ,,
Margarine ,,	6,164 ,,	Cod-fish ,,	656 ,,

If the amount of proteid obtained be taken as the standard, one gets such results as these:—1 lb. of proteid in peas costs 7d.; oatmeal, 7½d.; bread, 1s. 6d.; milk, 2s. 2d.; beef, 2s. 8d.

A glance at the tables will show that both as regards the total number of Calories yielded and the amount of building material obtained, the vegetable foods are very superior to animal products. Amongst the cheapest of the latter in both respects are cheese and milk.

It must be clearly realised that the maxim "cheap and nasty" does not apply to foods. High price and high nutritive value are not synonymous. The price of a food in the market is determined more by such considerations as flavour and rarity than by chemical composition or digestibility. Thus a pound of cod at threepence yields just as much nourishment as a similar quantity of sole at eighteenpence; and common arrowroot at fourpence is of the same chemical and physiological value as Bermuda at two shillings.

ANIMAL FOODS

1. *Meat.*—The flesh of animals, which constitutes meat, consists of muscle fibres held together by connective tissue. The fibres vary in length and thickness. If they are long and coarse, as in the leg of a crab, the meat is less easily digested than when the fibres are shorter and more delicate, as in the breast of a chicken. The fibres contain the muscle plasma, or "juice" of the meat, the chief ingredients of which are water, the proteid called myosin, a certain amount of hæmoglobin, certain "extractive" bodies, the best known being creatin, and some mineral salts, chiefly compounds of potassium and phosphoric acid. Rigor mortis is due to clotting of the myosin, and meat in that condition is tough. By and by acids are developed in the meat which soften the myosin, and the meat becomes tender again. Meat should, therefore, be eaten either before rigor mortis has supervened or after it has passed off.

The connective tissue of the meat consists of the substance called "collagen," which is converted into gelatine by boiling. The connective tissue is more abundant in old than in young animals, and the flesh of the former requires more cooking to convert the connective tissue into gelatine than that of the latter does. Embedded in the connective tissue is a certain amount of fat. In some kinds of meat, such as pork, the fat is abundant; in others, such as partridge and chicken, it is almost absent. In swimming birds, such as the duck and goose, too, the amount of fat between the fibres is relatively large. Fat so placed tends to hinder the access of the digestive juices to the fibres, and impairs the digestibility of the meat. It is well to forbid such meats to the dyspeptic. The chemical composition of the whole of the meat taken together is approximately this—

	Water.	Nitrogenous Matter.	Fat.	Mineral Matter.
Lean	76·5	21	1·5	1
Medium . . .	73·0	20·5	5·5	1
Fat	53·0	17	29·0	1

It will be observed that the fatter the meat the poorer it is in water. Fat and water are to a large extent mutually replaceable. Roughly speaking, three-fourths of ordinary meat consist of water. Of the nitrogenous matter, about 1 per cent consists of "extractives," and 2 to 3 per cent of albuminoids, such as collagen ; the rest consists of proteid.

The objects of *cooking* meat are (1) to develop its flavour ; (2) to improve its appearance by destroying its red colouring matter ; (3) to sterilise it and so increase its keeping properties, and diminish the risk of infection by any disease germs or parasites which it may contain. The principles to be observed in carrying out these objects are—(1) to heat the meat to a temperature sufficient to sterilise it, and destroy its red colouring matter without over-coagulating its proteid ; (2) to convert all the connective tissue as far as possible into gelatine ; (3) to effect these changes without removing from the meat any of the "extractive" matter to which it owes its flavour. In carrying out the first principle, it should be borne in mind that the red colouring matter of meat is destroyed, and the proteid coagulated, at a temperature of 170° Fahr., and that to go above this simply tends to harden the meat by over-coagulation. In striving as far as possible to convert all the connective tissue into gelatine, one must remember that meat is a bad conductor, and therefore the heat should be applied to it slowly, and given plenty of time to act upon the collagen. The third object—the conservation of the flavouring ingredients of the meat—is to be achieved by "sealing up" the meat by a brief exposure of the surface to a temperature sufficient to suddenly coagulate all the superficial layers of proteid, and so prevent the subsequent escape of the extractives. The methods by which these principles are successfully carried out in practice are described in the article on "Invalid Cookery," but it may be pointed out here that they are best attained by heating the meat (after the preliminary sealing) for a long time to a temperature not exceeding 170° Fahr., and that the method of stewing or braising is more in accord with correct principles of cookery than any other.

The digestibility of meat is not improved by any method of cooking. Indeed, it may be laid down as a general rule, to which there are few exceptions, that cooking diminishes the digestibility of animal foods and increases that of vegetable products. The truth of this as regards meat is borne out by the following observations of Jessen :—

3½ ozs. of raw beef are digested in 2 hours.
,, half-boiled beef are digested in 2½ hours.
,, wholly boiled beef are digested in 3 hours.
,, half-roasted beef are digested in 3 hours.
,, wholly roasted beef are digested in 4 hours.
,, raw mutton are digested in 2 hours.
,, raw veal are digested in 2½ hours.
,, raw pork are digested in 3 hours.

As has already been pointed out, meat is absorbed in the intestine very completely, only about 5 per cent of its dry matter being left behind.

The ease of digestion of raw meat and the smallness of the residue which it leaves in the intestine render it a food of special value in the treatment of certain affections of the stomach and bowels. It is best prepared by scraping the fibres away from connective tissue which holds them together by means of the back of a knife. The pulp so prepared may either be administered in the form of a sandwich, or, if that is inadmissible, it may be stirred into a small quantity of beef-tea. Celery salt is a useful flavourer. Cakes of such beef-pulp, lightly browned on the surface, form the basis of the "Salisbury" cure.

The composition of some of the internal organs of animals used as food is as follows:—

	Water.	Nitrogenous Matter.	Fat.
Kidney . . .	76·7	16·9	4·8
Liver . . .	69·8	21·6	5·4
Heart . . .	62·6	16·0	20·4
Lung . . .	79·7	16·1	3·2
Sweetbread . .	70·0	28·0	0·2
Tripe . . .	74·6	16·4	8·5

Owing to their dense structure, kidney, liver, and heart are difficult of digestion. The lungs contain a large amount of indigestible elastic tissue. Blood is also sometimes used as food, but it contains only 18-23 per cent of solids, and is not well absorbed. The presence of much nucleo-proteid in all of the above organs, except the heart and tripe, makes it prudent for the gouty to avoid their use, seeing that nucleo-proteid is a source of uric acid.

Jellies are derived from gelatine produced by boiling the collagen of connective tissue. Young tissues, *e.g.* calves' feet, are the most abundant source. Isinglass is a pure form of gelatine derived from the swim-bladder of the sturgeon, but it is rather expensive, and does not really go farther than ordinary gelatine. Commercial gelatines (*e.g.* Nelson's or Cox's) are as good as any other for feeding the sick. The nutritive value of jelly is considerable. Six ounces (a large helping) of good jelly yields 1⅜ ozs. of solids, of which about one-half is gelatine, the rest being sugar. This must be remembered in ordering jelly for diabetics. Gelatine being the best "proteid-sparer," jelly is an admirable food for febrile patients.

The "extractives" of meat are the main constituent of *soups* and *beef extracts.* The physiological action of these extractives may be stated thus:—(1) They yield no potential energy, and are therefore not foods. (2) They have no power of increasing the rate or force of the heart. Their "stimulating" effect on the circulation is to be attributed to the hot water with which they are taken. (3) It is doubtful if they have any stimulating action on the nervous system, but they appear to remove the *feeling* of fatigue. (4) They taste and smell agreeably, and are, therefore, powerful aids to digestion by helping to call forth a secretion of gastric juice. (5) When taken in excess they are apt to excite diarrhœa.

Hence clear soups, which consist of a solution of the extractives, have no nutritive value, but are useful at the beginning of a meal. The same may be said of beef-tea as ordinarily prepared. The following is the latest analysis of Liebig's extract (Kemmerich):—Water, 18 per cent; proteids and gelatine, 30 per cent; extractives, 25 per cent; mineral matter, 20 per cent; ether extract, 7 per cent.

It will be observed that it contains a considerable amount of soluble proteid, but as a teaspoonful of it only weighs 5 grms. it can never be taken in sufficient quantity to be of real value as a food. The same may be said of those preparations, *e.g.* Bovril, to which some of the powdered fibre of meat has been added. The large amount of salts which beef extracts contain is of very doubtful advantage, for there is no proof that the demand for salts is increased in fevers, or that the mineral matter so supplied can be made any use of.

Beef Juices are preparations which contain the coagulable proteid of meat. The method of making raw beef juice is described elsewhere. It usually contains about 5 per cent. of proteid. The amount in commercial beef juices varies from 20 per cent or more (*e.g.* Vitalia) to considerably less than 5 per cent (*e.g.* Valentine's), but the majority do not contain much more coagulable proteid than the home-made article. The large amount of extractives and mineral matter in the commercial juices is a positive disadvantage, as it renders it impossible to administer them in any considerable quantity; for that reason home-made juice is to be preferred. Beef juice is sometimes a useful article of diet in diarrhœa (especially in children), but it is difficult to administer anything like an adequate supply of nutriment in that form alone.

Fish.—There are two classes of fish—fat and lean. Lean fish is such as contains less than 2 per cent of fat (*e.g.* sole, flounder, whiting, haddock, cod, and trout). Of the fat fishes, mullet, halibut, and mackerel contain from 2-5 per cent of fat; the salmon, turbot, herring, and eel contain more than 5 per cent. The fat fishes are about equal in nutritive value to a similar weight of moderately fat beef; the lean fishes are poorer in nutrients than beef, and contain relatively more water and gelatine. All fish is poorer in "extractives" than meat, and for this reason fish is a less stimulating form of food than meat, and is sometimes preferable to the latter in some diseases, *e.g.* epilepsy. Lean fish is usually easily digested owing to the shortness of its fibres and the absence of fat; for this reason it is suited to invalids. Fat fish is difficult to digest, and the oil in it is apt to become rancid and irritate the stomach. It should be avoided by dyspeptics. The belief that fish contains much phosphorus, and is, therefore, peculiarly suited to brain workers is entirely unfounded.

Allied to fish are lobsters, crabs, and oysters. These have the following composition:—

Lobster and Crab.		
	Flesh.	*Body.*
Nitrogenous matter	19·17	12·14
Fat	1·17	1·14
Water	76·6	84·31

Oyster.	
Nitrogenous matter	6·2
Fat	1·2
Carbohydrate	3·7
Ash	2·0
Water	86·9

The coarseness of the muscle fibres in the flesh of the lobster and crab makes the limbs of these animals very difficult of digestion. Raw oysters are easily digested, but of low nutritive value owing to the high percentage of water. The nature of the carbohydrate material which they contain is doubtful.

Milk and its Products.—Cow's milk has the following approximate composition:—

Water	87-88 per cent.
Proteid	2-3 ,,
Fat	3½-4½ ,,
Sugar	4-5 ,,
Mineral matter	0·7 ,,

It fluctuates so greatly in composition, however, that one does not know within 30 per cent how much nourishment he is ordering in prescribing a given quantity of milk to a patient daily. Fat is the most variable element of all, and is usually taken as the criterion by which to judge milk. Good milk should contain 4 per cent.

The proteids of milk are caseinogen and lactalbumin, there being about one part of the former to seven of the latter in the milk of the cow. Caseinogen is a pseudo-nucleoalbumin. It yields no uric acid bodies, and no sugar on hydrolysis. This gives it a special value in gout and diabetes. Pure caseinogen is now prepared in the form of a flour (by the Protene Co.), and used in the preparation of diabetic bread and biscuits. "Nutrose" is caseinogen rendered soluble by being combined with soda.

The sugar of milk or lactose has the advantage of not being very sweet. It may thus be added to milk and other liquids, *e.g.* lemonade, to increase their nutritive value. Such addition is useful in the case of patients who are suffering from acute disease and are unable to swallow large quantities of nourishment. It is not fermented by yeasts, and may, therefore, be of use in cases of dilated stomach, but is readily changed into lactic acid by certain micro-organisms, and in that form is a common cause of diarrhœa in infancy.

The chief mineral matter in milk is calcium, one litre of it containing about a gramme and a half of lime. Some of the calcium is combined with citric acid, the rest occurs as phosphate. Milk contains very little iron, four or five pints being required to yield the amount of iron (10 milligrammes) required daily.

The germs which are so apt to be contained in milk can most easily be killed by boiling. Pasteurisation—which consists in keeping the milk at a temperature of 70° Cent. for twenty minutes — is sufficient to kill most disease germs, but does not destroy all spores nor the lactic-acid producing bacteria. It has the advantage of not affecting the taste of the milk. It may be carried out by placing the milk in stoppered bottles, and setting these in a deep saucepan of water heated to the necessary temperature.

The *digestibility* of milk is interfered with by the dense clot which it forms in the stomach. The density of the clot depends on (1) the amount of the casein; (2) the degree of acidity of the stomach contents; (3) the quantity of lime salts present. All of these factors have their influence lessened by dilution. Water may be employed for the purpose, but barley-water or lime-water are better. The former acts mechanically by entangling the particles of casein. Lime-water seems to have a specific power of preventing clotting apart from its alkalinity. One part to two of milk is sufficient to ensure a loose clot.

A pint of raw milk remains about 3½ hours in the stomach, sour or skimmed milk about 3 hours, and boiled milk about 4 hours.

Milk is not very completely absorbed. When it constitutes the sole diet only about 90 per cent of its potential energy is really available. Thus it leaves a larger residue in the intestine than many other foods (see Table II. p. 522). Children absorb it better than adults. Boiled milk is not *quite* so well absorbed as raw. The nutritive value of milk is high, but about 9 pints of it would be required daily by a man doing moderate work. 4-5 pints, however, are sufficient to maintain the nutrition of a sick person lying in bed. It contains too much water and too much proteid and fat in proportion to its carbohydrate, and is too expensive to justify one in regarding it as a perfect food.

Cream contains relatively much more fat than milk, but the actual amount varies greatly from 12 or 15 per cent in ordinary cream, up to 40 per cent or more in thick or separated cream. It contains as much sugar and proteid as a similar quantity of milk.

Butter has about 80 per cent of fat and a small amount of proteid, the remainder being water. Margarine, which is made from the more oily constituents of animal fat, has almost the same chemical composition, and is practically equal to butter in nutritive value.

Koumiss is produced from mare's milk by a combined lactic and vinous fermentation.

Kephir is a similar product prepared from the milk of the cow. In both the caseinogen is thrown down in a finely flocculent, and therefore easily digested, form, and is also partly peptonised; most of the sugar is converted into lactic acid, and a small amount of alcohol and a large proportion of carbonic acid gas produced. Three and a half quarts (a daily allowance) of koumiss yield 140 gms. of proteid, 80 of fat, and 140 of sugar, with a combined fuel value of 1918 Calories. These preparations present milk in its most easily digested form, and owing to the destruction of much of the lactose, are better in some cases of diabetes than ordinary milk.

Cheese consists of the casein and fat of milk, modified by the growth in it of various micro-organisms, to the bye-products produced by which the different varieties of cheese owe their distinctive flavour. The composition of the common cheeses is shown in the following table:—

COMPOSITION OF CHEESES[1]

Cheese.	Water.	Proteid.	Fat.	Ash.	Average Cost per lb.	Real Cost of of 1 lb. Nutriment.
					s. *d.*	*s.* *d.*
American	26·9	32·9	31	4·5	0 6	0 8⅕
Camembert	48·6	21·0	21·7	4·4	7d. each.	1 1⅗
Cheddar	31·9	33·4	26·8	3·9	0 9½	1 2
Cheshire	33·2	29·4	30·7	4·3	0 9½	1 2
Cream	32·0	8·6	35·9	1·5	...	...
Dutch	32·9	30·8	17·8	6·3	0 7	0 10⅖
Gloucester	31·9	36·7	24·7	4·4	0 9½	1 2
Gorgonzola	39·2	25·9	26·9	4·7	0 9	1 3
Gruyère	34·1	31·5	28·2	4·0	0 10	1 3
Parmesan	30·0	43·8	16·5	5·9	0 11½	1 4⅖
Roquefort	25·1	34·8	31·5	5·5	1 1½	1 6
Stilton	27·6	23·9	38·9	3·1	1 2	1 7

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[1] These figures are constructed by taking the averages of the analyses collected by Pearmain and Moor. Prices are those of the stores.

Cheese is a condensed and cheap form of animal food of high nutritive value, and specially adapted to supplement deficiency of proteid in the diet. Its density and richness in fat, however, render it difficult of digestion. This difficulty is best overcome by dissolving the cheese and then mixing it with some cereal food, *e.g.* macaroni. Solution can easily be brought about by adding to every quarter pound of the grated cheese as much bicarbonate of potash as will lie on a threepenny piece and stirring the whole with a little warm water or milk. The absorption of cheese in the intestine is fully equal to that of meat.

EGGS.—An ordinary hen's egg weighs about 2 ounces, of which about 12 per cent consists of shell, 58 per cent of white, and 30 per cent of yolk. The shell consists of carbonate of lime. The white and yolk have the following composition:—

	Water.	Proteid.	Fat.	Ash.
White	85·7	12·6	0·25	0·59
Yolk	50·9	16·2	31·75	1·09

The white consists of a solution of various proteids, the chief of which is egg albumin. Some, at least, of these yield a reducing sugar on hydrolysis.

The yolk contains a large amount of emulsified fat and a considerable proportion of lecithin. It contains also other organic compounds of phosphorus, amongst them a nucleo-proteid and an organic compound of iron. Indeed, yolk of egg is one of the richest food sources of iron that we possess. The fact that it contains so much fat and organic phosphorus seems to make yolk of egg of peculiar use to growing animals, and explains its value in the diet of children.

Experiments on their digestibility have shown that two lightly-boiled eggs remain in the stomach about 1¾ hours. If taken raw they remain rather longer, and raw eggs are not really more digestible than those which have been lightly cooked. Hard-boiled eggs remain about three hours. The absorption of eggs in the intestine is very complete, only about 5 per cent of residue being left. Hence eggs may be safely ordered in diarrhœa.

The nutritive value of one egg is equal to that of about half a tumblerful of milk. The potential energy yielded amounts to 70 Calories. Twenty eggs are required to supply the amount of proteid required daily.

VEGETABLE FOODS

The vegetable foods are distinguished from animal foods mainly by containing much more carbohydrate and considerably less proteid. The carbohydrate is chiefly in the form of starch, although some vegetable foods, *e.g.* fruits, contain sugar instead. The proteids met with belong mostly to the globulin class, and contain less carbon and more nitrogen than animal proteids, which is said to give them a lower nutritive value. The comparative poverty of vegetable foods in proteid renders it difficult to obtain a sufficient supply of nitrogen from them alone unless a large quantity is eaten, and the consumption of vegetable foods in quantity is inconvenient owing to their bulk. This bulkiness is due to the fact that most vegetable foods contain much cellulose and much water, for even the dry vegetable foods, *e.g.* the cereals and pulses, take up much water in process of being cooked. Hence the pure vegetarian has to choose between living on a minimum of proteid or consuming such a bulk of food as throws a considerable strain on the stomach and bowels. The adoption of the former alternative seems to diminish one's energy, as distinct from muscular strength, and to lower one's power of resistance to disease; while the latter course is apt to culminate in disorders of digestion. The disadvantages of a purely vegetable diet are increased by the inability of the intestines to absorb large quantities of vegetable proteid, as already pointed out, unless presented in very special forms. On the other hand, their comparative poverty in nitrogen adapts the vegetable foods for use in chronic renal disease and in gout, while their bulkiness is of value in habitual constipation by stimulating peristalsis, and in obesity by affording a maximum of quantity with a minimum of nutritive material.

The vegetable foods may be divided into the following classes:—

1. *Cereals.*—The composition of the most generally used of these is as follows:—

Composition of Cereals[1]

	Water.	Proteid.	Fat.	Carbo-hydrates.	Cellulose.	Mineral Matter.
Wheat	12·0	11·0	1·7	71·2	2·2	1·9
Oats	10·0	10·9	4·5	59·1	12·0	3·5
,, (hulled)	6·9	13·0	8·1	68·6	1·3	2·1
Barley	12·3	10·1	1·9	69·5	3·8	2·4
Rye	11·0	10·2	2·3	72·3	2·1	2·1
Maize	12·5	9·7	5·4	68·9	2·0	1·5
Rice (unhulled)	10·5	6·8	1·6	68·1	9·0	4·0
,, (hulled)	12·0	7·2	2·0	76·8	1·0	1·0
,, (polished)	12·4	6·9	0·4	79·4	0·4	0·5
Millet	12·3	10·4	3·9	68·3	2·9	2·2
Buckwheat	13·0	10·2	2·2	61·3	11·1	2·2

[1] The table represents the composition of the cereals in their crude form. The figures are compiled from a vast number of analyses, the data contained in the report on the composition of the cereals exhibited at the Columbian Exposition being freely used (U.S. Bull. 45). The proteid has been calculated from the nitrogen, using the factor 5·7 for all except barley, maize, and buckwheat, where the factor 6 was employed.

Composition of Products Derived from Cereals

	Water.	Proteid.	Fat.	Carbo-hydrates.	Cellulose.	Mineral Matter.
Wheat Meal	12·1	12·9	1·9	70·3	1·6	1·2
Fine Wheat Flour	13·0	9·5	0·8	75·3	0·7	0·7
Oatmeal	7·2	14·2	7·3	65·9	3·5	1·9
Rolled Oats	7·2	15·4	7·2	64·8	3·5	1·9
Barley Meal	11·9	10·0	2·2	71·5	1·8	2·6
Pearl Barley	12·7	7·4	1·2	76·7	0·8	1·2
Coarse Rye Flour	11·4	15·3	2·1	66·7	2·3	2·2
Finest ,,	11·2	6·7	0·9	80·0	0·8	0·4
Corn Meal	11·4	8·5	4·6	72·8	1·4	1·3
,, ,, (fine)	12·5	6·8	1·3	78·0	0·8	0·6
Buckwheat Flour	14·0	7·1	1·2	75·9	0·6	1·2
Rizine (flaked rice)	11·7	7·9	0·5	79·5	...	0·4

Of these wheat is mainly used in Europe, maize in America, and rice in the East. Maize is superior to wheat in fatty material, while rice is characterised by its small proportion of both proteid and fat. Oats are about equal to maize in fat and superior to wheat in proteid, and rank as the most nutritive of all cereals. A plateful of porridge is equal in nutritive value to three slices of bread. The chief product of wheat is, of course, bread. The following is the average composition of white and whole meal bread respectively:—

	White.	Whole meal.
Water	39	45
Proteid	6·5	6·3
Fat	1	1·2
Carbohydrates	51·5	45·3
Mineral matter	1	1·2

The greater moistness of whole meal bread causes it to be hardly at all superior to white bread chemically, while physiologically it is found to be not so well absorbed. It is therefore on no account to be preferred to white bread for ordinary use, though it is of value in cases of constipation. On the other hand, breads such as Hovis, which contain a large proportion of wheat germ, are peculiarly rich in proteid, of which they may contain as much as 9 per cent.

Biscuits, being almost free from water, are more nourishing than bread, three pounds of the former being equal to five pounds of the latter. They are also very easily digested.

Semolina is prepared from the central parts of hard wheat. It contains 10·6 per cent proteid.

Macaroni and *vermicelli*, which are prepared from good wheat flour by mixing into dough and then drying, have a similar composition.

Hominy consists of split maize and resembles the latter in its constituents, but has only ½ per cent of fat. *Cornflour* consists of practically pure starch prepared from maize.

2. The *Pulses* have the following composition :—

COMPOSITION OF PULSES

(*From the Means of many Analyses*)

	Water.	Proteid.	Carbohydrates.	Fat.	Cellulose.	Mineral Matter.
Green Peas	78·1	4·0	16·0	0·5	0·5	0·9
Dried ,,	13·0	21·0	55·4	1·8	6·0	2·6
Lentils	11·7	23·2	58·4	2·0	2·0	2·7
Horse Beans (dry)	13·1	25·5	50·9	1·7	5·5	3·3
Broad or Windsor Beans (dry)	8·4	26·4	58·6	2·0	1·0	3·6
French Beans (Haricots Verts)	89·5	1·5	7·3	0·4	0·6	0·7
Haricots (H. Blancs)	11·7	23·0	55·8	2·3	4·0	3·2
,, (cooked)	73·6	4·4	20·8 (and Cellulose)	0·5	...	0·7
Scarlet Runners (stewed)	91·12	1·7	3·7	0·3	2·9	0·3
Soy Beans	11·0	32·9	28·7	18·1	4·4	4·9
,, Bean Flour	9·3	39·5	28·2	13·7	4·0	5·3
Pea-Nuts	8·3	24·0	17·0	44·3	4·5	1·9

They are richer in nitrogen than any other vegetable foods. This they owe to the presence of a vegetable proteid called legumin or vegetable casein. Legumin forms insoluble compounds with lime salts, which is the explanation of the advantage of cooking the pulses in soft water.

The pulses are somewhat difficult of digestion in the stomach, and are apt to produce a feeling of repletion, even in small amounts. In the intestine they are prone to excite flatulence owing to their richness in sulphur. Lentils have least tendency in this direction. As a group the pulses are well absorbed provided they be given in a state of fine division.

Soy beans and pea-nuts are largely used in the preparation of diabetic foods owing to their poverty in carbohydrates. The preparation Revalenta Arabica consists mainly of Egyptian lentil flour.

3. The *roots* and *tubers* consist chiefly of starch, as the following table shows :—

COMPOSITION OF ROOTS AND TUBERS

	Water.	Proteid.	Carbo-hydrates.	Fat.	Fibre.	Ash.	Extrac-tives.
Potatoes	76·7	1·2	19·1	0·1	0·6	0·9	1·4
,, (boiled in skin)	73·8	...	...	...	...	...	...
Carrots	8·5	0·5	10·1	0·3	1·5	0·9	1·0
,, (cooked)	93·4	0·53	3·39	0·17	1·8	0·14	...
Turnips	90·3	0·9	5·0	0·15	1·8	0·8	1·1
,, (cooked)	97·25	0·32	0·65	0·06	1·2	0·32	...
Radishes	90·8	1·4	4·6	0·1	...	0·7	...
Beetroots	83·9	0·5	11·0[1]	0·1	3·0	0·9	1·0
,, (cooked)	94·8	0·44	2·83	0·06	1·3	0·3	...
Parsnips	80·1	1·4	14·1	1·0	2·1	1·3	...
,, (cooked)	97·28	0·22	1·46	0·29	0·72	0·12	...
Artichokes	79·8	2·3	14·5	0·3	2·0	1·0	...
,, (cooked)	91·6	1·8	4·6	0·08	0·9	0·61	...
Onions	89·1	1·6	6·3	0·3	2·0	0·6	...
Sweet Potatoes	72·9	1·6	22·5	0·5	1·8	0·7	...
Yams	79·6	2·2	15·3	0·5	0·9	1·5	...

[1] 10 per cent of Sugar.

Of the small amount of nitrogenous material which they do contain a large proportion is in the form of amido-bodies of little or no nutritive

COMPOSITION OF VEGETABLES

	Water.	Nitrogenous Matter.[1]	Fat.	Carbo-hydrates.	Mineral Matter.	Cellulose.	Fuel Value per lb.
							Cals.
Cabbage	89·6	1·8	0·4	5·8	1·3	1·1	165
,, (cooked	97·4	0·6	0·1	0·4	0·13	1·3	...
Cauliflower (head)	90·7	2·2	0·4	4·7	0·8	1·2	175
Sea Kale	93·3	1·4	...	3·8	0·6	0·9	...
,, (cooked)	97·95	0·4	0·07	0·3	0·2	1·1	...
Spinach	90·6	2·5	0·5	3·8	1·7	0·9	120
Vegetable Marrow	94·8	0·6	0·2	2·6	0·5	1·3	...
,, ,, (cooked)	99·17	0·09	0·04	0·2	0·05	0·37	...
Brussels Sprouts	93·7	1·5	0·1	3·4	1·3	...	95
Tomatoes	91·9	1·3	0·2	5·0	0·7	1·1	105
,, (cooked)	94·07	1·0	0·2	0·1	0·76	1·5	...
Greens	82·9	3·8	0·9	8·9	3·5	...	275
Lettuce	94·1	1·4	0·4	2·6	1·0	0·5	105
,, (cooked)	97·2	0·5	0·16	0·5	0·4	0·9	...
Leeks	91·8	1·2	0·5	5·8	0·7	...	150
Celery	93·4	1·4	0·1	3·3	0·9	0·9	85
,, (cooked	97·0	0·3	0·06	0·8	0·5	1·0	...
Turnip-Cabbage	87·1	2·6	0·2	7·1	1·5	1·3	145
Rhubarb	94·6	0·7	0·7	2·3	0·6	1·1	105
Macedoine (tinned)	93·1	1·4	...	4·5	1·0	...	110
Watercress	93·1	0·7	0·5	3·7	1·3	0·7	...
Cucumber	95·9	0·8	0·1	2·1	0·4	0·5	70
,, (cooked)	97·4	0·5	0·02	0·7	0·2	0·9	...
Asparagus	91·7	2·2	0·2	2·9	0·9	2·1	110
Salsify (cooked)	87·2	1·2	0·08	9·0	0·3	2·2	...
Endives	94·0	1·0	...	3·0	0·8	0·6	...
Savoys	87·0	3·3	0·7	6·0	1·6	1·2	...
Red Cabbage	90·0	1·8	0·19	5·8	0·7	1·2	...
Sauerkraut	86·3	1·5	0·8	4·4	7·0	...	...

[1] Probably only about a half of the nitrogenous matter consists of proteid.

value. Hence they are too poor both in proteid and in fat to be fitted to constitute the main part of any diet.

It is worth remembering that potatoes contain considerably less starch than bread, and may consequently be allowed in larger proportion than the latter to cases of mild diabetes.

Tapioca, *sago*, and *arrowroot* are all preparations containing about 88 per cent of starch, and practically no nitrogen. Alone they are of limited nutritive value, but are useful additions to foods, such as milk, which are richer in proteid. They rank amongst the most completely absorbed of all foods, which justifies their employment in cases of diarrhœa.

4. *Green vegetables* are of very low nutritive value, as is borne out by the foregoing table representing their composition.

They owe their chief importance as regular articles of diet to the alkaline salts, especially of potash, which they contain. These are excreted in the form of carbonates in the urine, and for that reason green vegetables should be freely consumed by patients with a tendency to gravel. Their richness in cellulose makes them difficult of digestion, but useful as sup-

COMPOSITION OF FRUITS

	Water.	Proteid.	Ether Extract.	Carbo-hydrates.	Ash.	Cellulose.	Acids.
Apples	82·5	0·4	0·5	12·5	0·4	2·7	1·0
,, (dried) . . .	36·2	1·4	3·0	57·6	1·8	...	...
Pears	83·9	0·4	0·6	11·5	0·4	3·1	0·1
Apricots	85·0	1·1	?	13·4	0·5	...	...
Peaches	88·8	0·5	0·2	5·8	0·6	3·4	0·7
Greengages . . .	80·8	0·4	...	14·4	0·3	4·1	...
Plums	78·4	1·0	?	20·1 (and Cellulose)	0·5	?	?
Nectarines . . .	82·9	0·6	?	15·9	0·6	...	...
Cherries	84·0	0·8	0·8	10·0	0·6	3·8	...
Gooseberries . . .	86·0	0·4	...	8·9	0·5	2·7	1·5
Currants (red, black and white)	85·2	0·4	...	7·9	0·5	4·6	1·4
Strawberries . . .	89·1	1·0	0·5	6·3	0·7	2·2	...
Whortleberries (= Blaeberries or Bilberries) .	82·4	0·7	3·0	13·5	0·4	...	...
Blackberries . . .	88·9	0·9	2·1	7·5	0·6	...	...
Raspberries . . .	85·8	1·0	?	12·6	0·6	...	...
Cranberries . . .	88·5	0·5	0·7	10·1	0·2	...	...
Grapes	79·0	1·0	1·0	16·0	0·5	2·5	...
Melons	89·8	0·7	0·3	8·6	0·6	...	...
Water Melons . . .	92·9	0·3	0·1	6·5	0·2	...	...
Bananas	74·0	1·5	0·7	22·9	0·9	0·2	...
Oranges	86·7	0·9	0·6	9·7	0·6	1·5	small quantities
Lemons	89·3	1·0	0·9	8·3	0·5	...	...
Pineapples . . .	89·3	0·4	0·3	9·7	0·3	...	...
Dates (dried) . . .	20·8	4·4	2·1	65·7	1·5	5·5	...
Figs (dried) . . .	20·0	5·5	0·9	64·0	2·3	7·3	...
,, (fresh) . . .	79·1	1·5	...	18·8	0·6	...	...
Prunes (dried) . . .	26·4	2·4	0·8	68·9	1·5	...	...
,, (fresh) . . .	80·2	0·8	?	18·5	0·5	...	...
Currants (dried) . .	27·9	1·2	3·0	65·7	2·2	...	...
Raisins	14·0	2·5	4·7	74·7	4·1	...	...

Composition of edible parts alone represented. Where cellulose is not given it is included with carbohydrates.

plying "intestinal ballast" in cases of constipation. They contain so little carbohydrate that they may be used, with very few exceptions, even in severe cases of diabetes. Cauliflowers being more easily digested than any other form of vegetable, may be advantageously recommended to the dyspeptic.

5. The *fruits* form a large class, whose composition is approximately as shown in table, p. 533.

They may be conveniently divided into "flavour fruits" and "food fruits." The former include all the berry class, and are chiefly eaten for the sake of their agreeable flavour. Like the green vegetables, however, they are also of service by reason of the vegetable salts of potash which they contain. The "food fruits" are represented by the fig, banana, date, and other dried fruits. They contain a large proportion of carbohydrate in the form of sugar, which gives them a quite considerable nutritive value, and are used in many countries, *e.g.* Egypt, in place of the cereals. It should be remembered that the chief sugar met with in fruits is lævulose, and that the latter is better utilised by diabetics than any other form of carbohydrate.

The *nuts* form a class by themselves, and are characterised by great richness in fat, as the following table shows:—

Composition of Nuts

	Water.	Proteid.	Fat.	Carbo-hydrates.		Cellulose.	Mineral Matter.
Chestnuts (fresh)	38·5	6·6	8·0		45·2		1·7
,, (dried)	5·8	10·1	10·0		71·4		2·7
Walnuts (fresh)	44·5	12·0	31·6	9·4	...	0·8	1·7
,, (dried)	4·6	15·6	62·6	7·4	...	7·8	2·0
Filberts and Hazels (fresh)	48·0	8·0	28·5	11·5	...	2·5	1·5
,, ,, (dried)	3·7	14·9	66·4	9·7	...	3·2	1·8
Sweet Almonds	6·0	24·0	54·0	10·0	...	3·0	3·0
Pistacio Kernels	7·4	21·7	51·1	14·0	...	2·5	3·3
Cocoa Nut (fleshy part)	46·6	5·2	35·9	8·4	...	2·9	1·0
,, ,, (dried)	3·5	6·0	57·4		31·8		1·3
,, ,, (milk)	90·3	0·5	...		[illegible]·0		...

Their richness in fat and cellulose, and the compactness of their structure, render them difficult of digestion. Being comparatively poor in carbohydrates they are largely used in the manufacture of diabetic foods.

6. The *fungi*, *lichens*, and *algæ* are a small and unimportant group of vegetable foods. The first-mentioned are represented by the mushroom and truffle, which have the following composition:—

	Mushroom.	Truffle.
Water	93·7	73·0
Proteid	2·2	6·1
Fat	0·3	0·6
Carbohydrate	1·2	10·2
Cellulose	1·1	6·4
Ash	0·3	2·0

These are very indigestible in the stomach—so much so, indeed, that the symptoms of indigestion which they are apt to produce have often been mistaken for poisoning. They are also very imperfectly absorbed, and cannot rank as foods of any value. The algæ are represented by Irish, and the lichens by Iceland moss. These have the following constituents (Church):—

	Irish Moss.	Iceland Moss.
Water	18·8	10·0
Proteid	9·4	8·7
Mucilage	55·4	70·0
Cellulose	2·2	3·5
Ash	14·2	1·5

The "mucilage" consists mainly of lichenin—a carbohydrate-containing material of doubtful nature. It certainly yields a reducing sugar on hydrolysis. The nutritive value of these substances is very low, for a teacupful of good Irish moss jelly contains only about a shilling's weight of solid matter. Decoctions of them make pleasant demulcent drinks, but that is the limit of their usefulness in the sickroom.

MINERAL CONSTITUENTS OF FOOD. — About 5 per cent of the body weight consists of ash. Mineral matters are therefore to be regarded as tissue-builders, and as such are essential to life, and as a matter of fact it is found that death ensues in a few weeks if no inorganic materials are supplied in the food. The chief mineral matters required are sodium and potassium, calcium and magnesium, iron, phosphorus, sulphur, chlorine, and traces of such substances as manganese and silica.

It is impossible to say exactly how much of any or all of these is required daily, for the reason, amongst others, that several of the mineral ingredients of the body are excreted by the large intestine, and one can, therefore, never distinguish those inorganic constituents of the fæces which have simply not been absorbed from those which have been absorbed and again excreted. An ordinary mixed diet, however, contains about 20 grammes of mineral matter, and that may safely be regarded as more than a sufficient supply. Further, it may be asserted that we know of no disease which is produced by an excess of mineral ingredients in the food. and of almost none, with the exception, perhaps, of scurvy, which is due to their deficiency.

Potassium is most abundantly represented in vegetable, and *sodium* in animal foods. The foods richest in *lime* are milk, eggs, cereals, and a few vegetables, such as radishes, asparagus, and spinach. Meat, fruits, and potatoes are poor in lime. It has, therefore, been recommended that one should forbid the former class and order the latter to patients who are suffering from atheroma. The utility of such a proceeding is, however, very doubtful.

About 10 milligrammes of *iron* are contained in an ordinary mixed diet (Stockman). The foods richest in that metal are yolk of egg, potatoes, bread, oatmeal, and rice. Milk is one of the poorest of all foods in iron, but five pints of it contain the requisite 10 milligrammes. The comparatively small amount of iron contained in ordinary food renders it impossible to look to dietetic means alone as affording a sufficient method of treating anæmias.

Phosphorus seems to be of special value in aiding the formation of new tissues. It is probable, however, that if it is to be available for that purpose it must be supplied in organic combination, and not simply as inorganic phosphates. The yolk of egg and tissues rich in nucleins, *e.g.* the roe of fish, sweetbreads, etc., are the richest sources of organic phosphorus. The importance of phosphorus as a brain food is probably overrated.

Sulphur is almost wholly taken into the body in the form of proteids, little or no inorganic sulphur being contained in an ordinary diet.

Chlorine is largely consumed in the form of common salt, of which about 10 grammes are usually added to the diet in addition to that which ordinary foods already contain. That the actual addition of salt to the diet is unnecessary is proved by the experience of those who live without it altogether. On the other hand, the evil effects attributed to its use are entirely imaginary. It is an undoubted aid to digestion, and, provided enough water be supplied as well, tends to limit rather than increase tissue waste.

BEVERAGES

The use of alcoholic beverages has been discussed under "Alcohol." We may therefore confine ourselves here to the consideration of water and the alkaloidal beverages (tea, coffee, and cocoa).

Water makes up about two-thirds of the body. Like the mineral matters, it is thus an important tissue builder and essential to life, death resulting in a few days if it be cut off. About 2½ pints of water should be consumed daily in addition to that already contained in ordinary solid food. Obviously, however, this quantity will vary greatly with such conditions as temperature and work. It is almost impossible, by the free drinking of water, to increase the amount of it in the blood, nor can one, by limiting the supply of water, render the blood appreciably poorer in it. On the other hand, it seems probable that the habitual ingestion of an excess of fluid may ultimately produce a more watery condition of the tissues, and if these be already water-logged a diminution of fluids in the diet may cause the passage of water from them back into the blood. This may explain the favourable results sometimes obtained by limiting the supply of fluids in cardiac dropsy.

Water is not absorbed in the stomach, but leaves it—if half a pint be taken—in the course of about half an hour. Even large quantities of water do not appreciably delay the digestion of food in the stomach, while in the intestine it probably aids somewhat the process of absorption. The rapid passage of water through the stomach makes it a peculiarly dangerous vehicle of disease. If the source of the water be at all suspicious, therefore, it is best to boil it, and re-aerate it in a gazogene, for no filter is really reliable for domestic use.

Water does not increase tissue waste, but merely washes out the waste products. For this reason its free use in gout, renal disease, and diabetes should be encouraged, and care should be taken to see that the patient gets enough of it in fevers, and if the pylorus is obstructed a due provision of water should be ensured by the use of saline enemata. Otherwise there is danger of the supervention of toxic symptoms from the retention of waste products.

Aerated waters are more favourable to digestion than ordinary water, for the carbonic acid gas which they contain acts as a stimulant to the stomach. This gas is rapidly absorbed through the stomach wall, hence it is well to avoid aerated waters in marked cyanosis. They should also be forbidden to patients in whom sudden distension of the stomach may be dangerous, *e.g.* cases of gastric ulcer or cardiac embarrassment.

TEA, COFFEE, AND COCOA.—*Tea* contains the alkaloid theine, along with some volatile oil, tannic acid, gummy matters, etc. Indian and Ceylon teas are richer in all the former ingredients than China teas, and green tea is richer than black. An ordinary teacupful of tea infused for five minutes will contain at least one grain of tannic acid, and one or two grains of theine. The longer the tea is infused—up to about half an hour—the more tannic acid will it contain. If one wishes to minimise the amount of tannic acid, one should use China tea and infuse it for four minutes only. The addition of milk also aids by precipitating the tannic acid.

Coffee contains caffeine (which is identical with theine), tannic acid, and an oily substance called caffeol, besides other less important ingredients. A teacupful of black coffee has about the same amount of caffeine and tannic acid as a similar quantity of tea. "French" coffee consists usually to a large extent of roasted chicory, the chief ingredient of which is caramel.

Cocoa contains the alkaloid theobromine, but in small amount; it is closely allied to caffeine. In addition it has (in its ordinary dietetic form) about 26 per cent of fat, 12 per cent of proteid, and 25 per cent or so of carbohydrates, besides a considerable quantity of ash (about 4 per cent).

All of these beverages have a retarding influence upon digestion, the effect of tea being greatest. Coffee is apt to irritate the stomach by its caffeol. It should therefore be forbidden in cases of gastric catarrh. Cocoa has least influence on the digestion of other foods, but the large amount of fat which it contains is apt to make it disagree.

The only member of the group which has any claims to be regarded as a food is cocoa. Seeing, however, that only about 10 grammes of cocoa are used to make a breakfast cupful of the beverage it cannot seriously aid in general nutrition. Indeed, seventy-five such cups would be required to supply the amount of potential energy required daily.

Tea and coffee are chiefly useful in virtue of the caffeine and volatile oil which they contain. These act as stimulants to the nervous system, and to some extent to the heart also, removing the sense of fatigue and increasing reflex action. The fact that they are usually taken hot no doubt contributes largely to their stimulating effects. The effects of tea and coffee on general metabolism is in the direction of increasing rather than of retarding body-waste.

These beverages are useful in nervous exhaustion and in alcoholic coma, they might also be used more largely than they are in fever. On the other hand they should be avoided or used with great moderation by "nervous" people, and by those suffering from sleeplessness or palpitation. Their effects on digestion sometimes renders it necessary to forbid their use in dyspepsia, although it is probable that the digestive disturbance attributed to the use of these beverages has been greatly exaggerated.

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UNDER the vague term digestion all these processes may be included to which the food substances are subjected, and the resultant changes which the various food constituents undergo in order to prepare them for assimilation by the tissues. It therefore includes a manifold series of processes all tending towards the same point, namely, the transformation of substances, which in their original state could not be made use of by the cell, into pabulum which can easily be taken up, assimilated, and used for the nutrition of the tissues. The most marked alterations which the food undergoes are brought about by secretions which special glands elaborate, the action being an extracellular one, and carried out mainly by the action of enzymes. These non-living ferments have specific actions on one or other of the food constituents. But there is an intracellular as well as an extracellular digestion, where the changes which the assimilated material undergoes, can only take place in the living cell. Our knowledge of these intracellular changes is still very limited, and in the vast majority of cases we can at most say that certain constituents of the blood-plasma are taken up by a certain group of cells, and that they leave it in other forms of combination (see "Blood"). Most of our knowledge of such processes has been derived from the histologist, although the information so obtained is rather indefinite, owing to the great difficulties encountered in comparing structural with chemical alterations. It is almost impossible to know definitely whether there be at the root of all cellular digestion (*e.g.* auto-digestion of organs) the action of enzymes, and it is also difficult to be certain whether the ferment has been formed in the cells of a particular organ, or transmitted to the latter as a zymogen from other parts. The decomposition of the food principles into smaller molecules takes place gradually, the final products of metabolism being formed in the tissue cells where these bodies, which are circulating in the blood, and are required for the special work of the cell, are selected, and are in part used up to furnish energy for its specific life processes, in part also stored up either for its own use or that of other cells. The final transformations result always in the production of work and heat, and those products which cannot be used further by the organism are excreted, their further decomposition taking place outside the animal body. One of the most marvellous things in the cell life is the peculiar stamp which each cell or group of cells gives to its work, although the ultimate principles, whose decomposition affords the energy necessary for the particular rôle of the tissue, are the same. What is ordinarily understood by the term digestion, however, is the series of changes which the food undergoes in the alimentary canal, because it is there that the most marked alterations take place, and it is with pathological disturbances from this side that the physician has to deal in cases of disordered digestion. The food is subjected in the alimentary canal to processes of two great types—mechanical and chemical. They are not, however, independent of one another, and both are under the influence of the nervous system. In order to prepare the food for absorption, it is necessary to break it down and transform it into material which can pass into the blood or lymph.

SALIVARY DIGESTION

Salivary digestion has two functions to perform; in the first place, the preliminary preparation of the food-stuffs for digestion with the saliva, gastric, pancreatic secretions, etc.; and secondly, a specific action, the transformation of insoluble carbohydrates into dextrins and a soluble sugar, maltose.

The saliva is formed from the secretions of the parotid, submaxillary and sublingual glands chiefly, with the addition of a mucous fluid from the buccal glands. It always contains suspended material, for example, epithelial squames from the palate, salivary corpuscles, leucocytes from the tonsils, etc.; but when freshly secreted the fluid is clear, rapidly becoming cloudy, however, owing to a precipitation of calcium carbonate from the removal of the carbonic acid, which kept it in solution. Normally it reacts slightly alkaline, but it may be neutral or slightly acid from lactic acid fermentation set up by organisms acting on food remnants which have adhered to the teeth. Its stringy-like consistence is due to admixture with mucus. The specific gravity varies from 1·002-1·008. The principal constituents are water, salts (especially chlorides and phosphates of sodium, sulphocyanates), mucin, a diastatic ferment termed ptyalin, and a trace of albumin. Oxygen, carbonic acid, and a fairly large quantity of nitrogen are present. The most important constituent is the *enzyme, ptyalin*, which is produced by the serous salivary glands, although it is not present in the cells in the active form, but rather as a forerunner or zymogen. The activity of this enzyme is destroyed by warming the saliva to a temperature above 70° C. One may say that it is most active in a slightly acid medium (due to organic acids) at a temperature between 35° and 37° C. The action takes place rapidly, and is best studied by mixing saliva with mucilage of starch, and keeping the mixture at the above-mentioned temperature. The fluid first becomes clearer, and then the action runs through the later stages so rapidly that on examination, even after a few minutes have elapsed, the final products of the digestion have been formed. The first body that is formed is soluble starch or amidulin, which takes up water, and is split up into a form of dextrin—erythrodextrin—and maltose. From the former another dextrin—achröodextrin—and maltose are formed. Erythrodextrin forms a coloured compound with iodine (of a red tint), while achröodextrin does not. In addition to maltose there is probably formed a small quantity of another sugar, isomaltose, and perhaps glucose. Sulphocyanates are by no means always present in saliva, only their presence or absence has no acknowledged pathological significance.

The most important *pathological constituents* are lactic acid in diabetes (never glucose), uric acid in gout, and urea in various nephritic conditions. Certain drugs may be excreted by way of the saliva, *e.g.* mercury, iodine, bromine, etc. Blood or pus may be present in inflammatory conditions of the mouth.

Nervous Mechanism of Secretion.—As a rule no saliva passes out from a salivary fistula unless there be chemical or mechanical stimulation of the surrounding parts; but the mucous secretion seems to be constantly produced. In man, however, even the sight or smell of food may set up salivation. If the glands be active, stimulation of the special taste nerves, or the sensory nerves of the mouth will at once produce a flow of saliva. Mastication greatly increases the flow, owing to a more frequent stimulation of the nerve endings in the buccal mucous membrane by the substances which are undergoing solution or maceration. Severe irritation of the stomach, as in emesis, will produce salivation by means of the stimulation of the vagus. There are two great sets of nerve fibres passing to the salivary glands, cranial and sympathetic, both containing secretory fibres, and in addition the former contains vaso-dilator, and the latter vaso-constrictor fibres. The same nerves pass to the submaxillary and sublingual glands, while the parotid has a special supply. It is unnecessary to describe in this place the course of these fibres, suffice it to say that the cranial supply of the two first-mentioned glands is the chorda tympani branch of the facial passing through the lingual branch of the fifth nerve, while that of the parotid is from the glosso-pharyngeal, through Jacobson's nerve, the small superficial petrosal, the otic ganglion, and the auriculo-temporal branch of the fifth nerve. All three glands have a supply from the cervical sympathetic. On stimulation of the cranial branches there is an abundant watery secretion, poor in solids, while after stimulating the sympathetic, a sparse secretion, rich in solids and of stringy-like consistence, is produced. Reflex stimulation from the mouth under ordinary conditions affects simply the cranial nerves, and interruptions in this reflex may occur, so that stimulation of the gustatory nerves will not produce an effect. During secretion the temperature of the saliva rises slightly above that of the blood in the carotid, and its pressure may also rise above that of the carotid. During active secretion an electrical current is produced in the gland, "the current of action." Nicotin and atropin paralyse secretion, the former affecting probably the nerve terminations around peripheral ganglia, the latter the final terminations around the secreting acini.

The morphological changes in the salivary glands during secretion are of importance. It is customary to speak of the parotid as a true serous gland, the sublingual as a mucous, and the submaxillary as a mixed gland; but the human sublingual gland in all probability produces a mixed secretion, because the crescentic cells lying at the periphery of the mucous acini are serous in type. Serous cells, as, for example, those of the parotid, contain at the beginning of salivary digestion numerous granules in the zone next the lumen of the acinus, rendering the cell cloudy in appearance, while the peripheral part around the nucleus is clearer. Gradually these albuminous granules are discharged, and the whole cell appears less opaque. The cells which secrete mucin appear clear and distended before discharge of their contents, while after secretion they are collapsed. Slowly mucinogen granules form for a little distance around the nucleus, and then, on taking up water, these swell and distend the free part of the cell. In all, the type of secretion is the same, material being taken up and stored in the outer part of the cells and then gradually transformed into mucin or albuminous material in the inner part and passed out as the true secretion of the acini. The part which the nucleus plays in the process is still doubtful.

The amount of saliva secreted during the day varies, on an average about $\frac{1}{2}$ to 2 litres. There may be a marked increase, as, for example, after mercury, potassium iodide, pilocarpine, and many vegetable irritants which stimulate the buccal mucous membrane. It may also be increased in many cases of nervous shock, while it is often greatly decreased in febrile conditions, and may be absent altogether in hysteria. Pathological changes in the salivary glands will be referred to under their special headings.

GASTRIC DIGESTION

Gastric Digestion.—Gastric digestion is in part a mechanical process, the food substances being brought into a condition of finer subdivision by means of the movements of the stomach; in part a chemical one, the finely subdivided food substances being gradually converted into a more soluble form, and one therefore better fitted for absorption. Under normal conditions the process of digestion is unaccompanied by sensation.

It is perhaps best to consider first the chemical process of digestion, prefacing this with a short account of the *chemical composition of the gastric juice.*

Gastric juice, as obtained from a case of gastric fistula in the human subject by Schmidt, was found to be a colourless transparent fluid of a specific gravity of 1·0022-1·0024. On boiling it becomes faintly opalescent, but yields no precipitate. Under normal conditions it appears not to be formed except as the result of stimulation; but even in a state of hunger the stomach is rarely found quite empty. The quantity present varies considerably. Anything above 50 cc. may be regarded as pathological. Many observers hold that the small quantity of 10-20 cc. normally found during hunger has been secreted as the result partly of stimulation due to the swallowing of saliva, partly of the mechanical stimulation arising from the introduction of the gastric sound. The degree of acidity varies considerably even in the same individual; but ·2 per cent may be regarded as a normal average.

In the dog, from which gastric juice can be obtained in considerable quantities, and free from the constituents of the saliva, and in which its composition has been much more carefully studied than in man, it is a colourless, odourless, transparent, strongly acid (containing about ·5 per cent hydrochloric acid), and actively peptic fluid of an average specific gravity of 1·0047. It may be kept an indefinite time without showing signs of putrefactive decomposition. It is slightly dextrorotatory, and gives the usual proteid reactions. On being cooled to 0° C. a fine precipitate of great peptic activity forms. The chief organic constituents are the enzymes, pepsin, rennin, and possibly invertin, along with traces of mucin and proteid convertible into albumose and peptone on standing. The inorganic constituents are chlorides of sodium, potassium, calcium, as well as traces of magnesium and iron. Phosphates are also present in very small quantity.

The free acid of pure gastric juice is hydrochloric, the most important proof of this being that the quantity of chlorine present is greater than the chemical equivalent of the total inorganic bases.

Conditions influencing the Secretion Sources and Mode of Formation of the Constituents of the Gastric Juice.—The secretion is normally intermittent. It may be induced (1) by psychical stimuli, *e.g.* the sight, or even the thought of food, (2) reflexly by stimulation of the nerves of taste, even where no food is allowed to enter the stomach, as in the pseudo-feeding of dogs after the establishment of a Pawlow fistula, and lastly by the entrance of food or other substances into the stomach.

Simple mechanical stimulation of the gastric mucosa produces only a slight local secretion. It is unlikely, therefore, that the normal secretion is at all largely dependent on mechanical irritation. Digestible solid substances mixed with saliva on their entrance into the stomach induce an immediate slight local secretion due to mechanical irritation. After about 15 minutes there follows a second secretion of gastric juice in much larger quantity from the whole surface of the gastric mucosa. The latter secretion is believed to follow as a result of the absorption of the small quantities of albumose and peptone produced by the action of the gastric juice secreted in consequence of the mechanical stimulation.

In the earlier stages of digestion the acid of the gastric juice is neutralised by the alkali of the food and of the saliva. For a variable period (three quarters of an hour according to Van d. Velden) free hydrochloric acid cannot be detected, the hydrochloric acid entering into loose combination with the albumoses, peptones, and albumins (to form acid albumin in the latter case) as quickly as it is secreted. During this period—the amylolytic stage of gastric digestion—the ptyalin of the saliva continues to exert its action upon the carbohydrate material of the food. As digestion proceeds the acidity of the juice gradually increases.

The hydrochloric acid and pepsin of the gastric juice are produced by different cells in the gastric glands. In the cardiac region of the stomach two varieties of cells are found, the centrally placed, chief or adelomorphous cells, and the parietal, delomorphous or oxyntic cells. In the pyloric region one kind of cell alone is present, and that resembles the central cells of the cardiac region very much more closely than the parietal ones. The pyloric secretion, when obtained free from the cardiac one, is found to be alkaline, and when acidified with hydrochloric acid it possesses peptonising power. The amount of pepsin, which it contains, is, however, much smaller than that of the cardiac region. The secretion of the cardiac region, on the other hand, contains both pepsin and hydrochloric acid. These, along with other facts, indicate the parietal cells to be the producers of the hydrochloric acid, and the central cells to be the originators of the pepsin. The site of the formation of the rennin ferment or its precursor is uncertain. With regard to the histological appearances of the gland cells, only a few words are necessary. The central cells in the resting condition are filled with distinct granules, which, during digestion, decrease in number. Typically, the granules are situated chiefly in the inner or luminal parts of the cells, the outer zone of the cells showing a complete absence of granules. In other rarer cases the granules are found scattered throughout the whole body of the cell, and the decrease is a general one.

In the ovoid parietal cells the granules are much smaller, and the changes not so distinct.

The granules in the central cells consist not of fully formed pepsin, but of a precursor or zymogen, which, under the influence of dilute acids, and also during active secretion by the cell, is converted into pepsin. To give a full proof of this statement would take up too much space. The salient points of difference between the zymogen (pepsinogen) and the enzyme, pepsin, are that the former is insoluble in glycerine, not easily destroyed by alkaline solutions, and possesses no digestive activity until converted into pepsin by dilute acids. The latter, on the other hand, is soluble in glycerine, very easily destroyed by dilute alkalies, and is an active digestive enzyme.

The distinction of rennin from its zymogen is founded on similar facts.

The Nervous Mechanism of Secretion.—The secretory nerves are the vagi. In a dog, in which a gastric fistula had been established and the œsophagus cut through, the eating of food induced an immediate secretion of gastric juice, accompanied by reddening of the gastric mucosa owing to vascular dilatation. This reflex secretion ceased to occur after both vagi had been divided. Stimulation of the peripheral ends of the vagi by slowly repeated induction shocks also produced secretion.

In a human subject in whom the lumen of the œsophagus had become closed so that a gastric fistula had to be formed, the chewing of food, none of which entered the stomach, produced a copious flow of gastric juice.

The Motor Mechanism of Gastric Digestion.—The stomach is usually closed at both its orifices, the cardiac and pyloric openings, by the contraction of the sphincter-like, circularly arranged muscle fibres. The cardiac orifice is opened during each act of swallowing at the close of the peristaltic contractions of the œsophagus, and the pylorus is opened from time to time to allow the passage of the liquified food material or chyme into the intestine. During the process of digestion the stomach walls are in continual peristaltic movement, thus ensuring the thorough admixture of the food with the gastric secretion, and also permitting the mechanical subdivision of the food masses. By these contractions the food material is propelled in two directions, the circumferential portions of the food passing towards the pylorus, while the central portions pass towards the cardiac orifice. This circulatory movement brings the digestive secretion into intimate contact with the food substances. The muscular movements are most active in the region bordering upon the pylorus (antrum pylori), which for this reason is sometimes separated by an oblique furrow from the remainder of the stomach. In the early stages of digestion the pylorus is firmly closed; but towards its termination the closure becomes much less firm, so that the contractions of the antrum pylori succeed in propelling the more fluid portions of the mass through the pyloric opening. Cold water is said by some to produce rapid opening of the pylorus.

The gastric movements are brought under the control of the nervous system by means of two sets of fibres. The vagi contain accelerator fibres, so that their stimulation induces peristaltic movement. Inhibitory impulses are transmitted through fibres, which in the dog leave the cord by the anterior roots of the fifth to the eighth thoracic spinal nerves. These fibres thence pass into the sympathetic system, have their cell stations in the cœliac ganglion, and ultimately reach the stomach by way of the splanchnic nerves.

The duration of digestion in health varies considerably according to the nature and quantity of the food taken, and also to individual idiosyncrasies. The normal limits lie between three and seven hours after a substantial meal, *e.g.* Riegel's test meal. A longer stay of food material in the stomach indicates motor insufficiency.

FUNCTIONS OF THE GASTRIC JUICE

Digestion commences in the stomach. In the mouth the food substances become mixed with the saliva, but their stay there is too short to allow of much digestive action taking place. The chief changes produced by the gastric juice are the following:—

1. The soluble nutriment, which has escaped solution by the saliva, is dissolved.

2. The ptyalin of the saliva converts the starch into dextrins and maltose. This action continues as long as there is no free acid present in the stomach.

3. The acid of the gastric juice brings into solution any earthy phosphates or carbonates introduced with the food.

4. The gastric juice, in virtue of its pepsin and hydrochloric acid, dissolves insoluble and coagulated proteids and their allies, converting them into bodies resembling globulins. Those proteids, which are already in solution, become chemically altered. The proteids undergo conversion into a body (acid albumin) precipitable on neutralisation, and not coagulable by heat. At a later stage bodies are formed which are not precipitable by neutralising the fluid; and as digestion proceeds the solubility of the bodies formed increases, and their precipitability by various chemical agents, *e.g.* saturation with neutral salts of the alkalies and magnesium, solutions of salts of the heavy metals, alcohol and strong mineral acids, diminishes. They also become more easily diffusible through animal membranes.

A chemical classification of the different products of gastric digestion has been founded upon these differences of solubility and precipitability. This classification is of considerable theoretical interest; but on account of the complexity and time-consuming nature of the methods employed for the complete separation of the different products, it has not, as yet, become of much importance in practical medicine. A very brief account of the most important facts must therefore suffice. The final product of gastric digestion is called peptone. The bodies representing the intermediate stages between it and acid albumin are called albumoses (or proteoses). The albumoses are all precipitable by saturation with ammonium sulphate, whereas peptone is not precipitable by this salt. Certain of the albumoses are also precipitable by saturation with sodium chloride or magnesium sulphate in neutral solution, and these have been named the primary albumoses. The remaining albumose is not precipitable by saturation with sodium chloride in neutral solution, and it is named deutero-albumose.

The pepsin and hydrochloric acid probably act as hydrolytic agents. This conclusion is supported (1) by the fact that the digestion of carbohydrates by the enzyme ptyalin is known to be hydrolytic; (2) by the fact that similar products to those of gastric digestion may be produced by boiling proteids with dilute mineral acids, which are known to act as hydrolytic agents; and (3) by the fact that peptone may be converted by certain dehydrating agents into bodies resembling native proteids.

With regard to the relative digestibility of different proteid and albuminoid bodies, the article on diet should be consulted.

Conditions affecting the rapidity of action of pepsin and hydrochloric acid.—The most favourable degree of acidity is about 0·2 per cent hydrochloric acid. Other acids may replace the hydrochloric, but all of them are much less efficient.

The most favourable temperature for the action of pepsin lies between 35° C. and 50° C. In neutral solution pepsin is destroyed by heating to 55° C.; but the addition of peptones to the solution raises its resisting power, so that a temperature of 60° C. becomes necessary for its destruction. In the dry condition it may be heated to a temperature of over 100° C. without losing its activity. Up to a certain maximum an increase in the quantity of pepsin accelerates digestion.

Numerous organic compounds, which in dilute solution destroy, or at least inhibit the activity of micro-organisms, exert little deleterious action on pepsin. Arsenious and salicylic acids, phenol, thymol, and chloroform in dilute solution only slightly retard peptic digestion. Neutral salts of the metals of the alkalies and alkaline earths lower the activity of pepsin. Alkalies and salts of the heavy metals rapidly destroy pepsin. It is of some importance to remember these facts in connection with the administration of drugs.

Accumulation of the products of digestion lessens greatly the rapidity of the action of pepsin. The duration of peptic digestion is thus greatly lessened by the simultaneous absorption of peptones by the stomach.

In virtue of the presence of the *rennin ferment* the gastric juice produces coagulation of the caseinogen of milk by converting it into casein. This action takes place in neutral solution prior to the appearance of free hydrochloric acid in the gastric contents. The caseinogen of milk is also precipitable as such by the acid of the gastric juice, and the precipitate is afterwards peptonised. The gastric juice splits up cane-sugar into dextrose and lævulose. This action is said by some observers to be due to a ferment *invertin*.

Glucose and lactose are also said to be split up into lactic acid by means of an enzyme, even when bacterial action is excluded. This enzyme, however, has not been isolated, and its existence is doubtful. The quantities of lactic acid so formed are, in any case, so small that the occurrence of appreciable quantities is certainly to be regarded as pathological.

The hydrochloric acid of the gastric juice, in addition to its digestive action, acts as a *germicidal agent*. When the secretion of hydrochloric acid is deficient, the growth of micro-organisms occurs much more readily. Amongst these organisms may be mentioned the bacterium lactis, which by its action on carbohydrates produces lactic acid. Bacterial decomposition of proteids is also liable to occur.

(1) *Examination of gastric contents.*—1. Methods used to obtain these.—The next point to consider is the *examination of the gastric contents.* It has already been mentioned that mechanical and electrical stimulation may be used to produce secretion of gastric juice. The most natural stimulus, however, is the introduction of digestible food material. The object of the examination of the gastric contents is to find out how the stomach fulfils its normal task of peptonising food material and propelling it into the intestines as the acid chyme. For this purpose a considerable variety of "test meals" has been employed.

The following are the more important ones:—

1. A test breakfast (Ewald and Boas), consisting of 70 gms. white bread and 300-400 cc. of weak tea or water.

2. A lunch (G. Sée), consisting of 100-150 gms. bread, 60-80 gms. of finely minced meat, and a large glass of water.

3. Riegel's test meal (given from 1-2 P.M.), consisting of a plate of soup (about 300 cc.), a beefsteak of 150-200 gms., potatoes 50 gms., and white bread 50 gms.

Klemperer, Bourget, Jaworski, and Gluzinski have also proposed test meals.

In considering the advantages and disadvantages of these meals, it will be found most convenient to take Ewald's breakfast and Riegel's dinner as types of the two extremes in quantity.

The chief advantages claimed for Ewald's method are the following:—

1. The composition of the meal is definite and simple.

2. The siphoning off of the gastric contents can be undertaken after a short and definite interval (1 hour).

3. Practically every patient is able to take the meal on account of its small size.

Riegel's meal, on the other hand, is somewhat indefinite and complex in composition. It is difficult to fix upon the most favourable interval of time after the meal, for the obtaining of the gastric contents, as it varies in individual cases, according to the degree of motor and peptic efficiency, from 2-6 hours. Usually the examination of the contents is undertaken 3½ hours after the meal. Lastly, the meal, on account of its large quantity, is not easily taken by all patients. Riegel's meal has, however, some advantages not shared by that of Ewald and Boas. In the first place, it lays a much greater demand upon the many-sided functional activity of the stomach, and therefore frequently gives one a better insight into what the stomach is really capable of performing. The results obtained by these methods do not always agree. The total acidity is usually found higher after Riegel's test meal than after that of Ewald and Boas. On the other hand, one must not omit to mention that in some cases, in which the production of hydrochloric acid is greatly diminished, *e.g.* in carcinoma ventriculi, free hydrochloric acid may be found after Ewald's test meal, whereas after that of Riegel a negative result of the examination is not infrequent. The explanation of this apparent contradiction lies in the fact that the large amount of proteid in Riegel's test meal is able to enter into loose combination with all the hydrochloric acid secreted. Another important advantage is that it may be employed in the same way as the similar meal advised by Leube for testing the motor efficiency of the stomach. For general use, if only one method be employed, probably Ewald's is the more convenient.

The gastric contents may be obtained by means of a soft rubber tube or sound about 75 cm. in length, and of variable diameter, 6-7 mm. being the average, to whose upper end a small glass tube (about 4 inches in length) is connected by means of rubber tubing. The lower end of the sound may be closed or possess a terminal aperture, but in all cases should have one large or several small (Ewald and Rosenheim) lateral openings. The presence of the lateral openings lessens the risk of obstruction by food particles. The sound, having been rendered aseptic, is placed in warm water. In syphilitic, tuberculous, or cancerous cases, separate sounds ought to be used for each class of case. The upper glass end is then put into a vessel, which may be held by the patient. The lower end of the sound is next introduced into the patient's mouth, and pushed as far back as the root of the tongue (for this manipulation the introduction of the operator's finger is unnecessary). If the patient now swallow once or twice the tube will glide into the upper end of the œsophagus, its descent into the stomach being afterwards aided by slowly and rhythmically pushing it onwards until about 45 cm. of the tube have been introduced.

For obtaining the gastric contents one of two methods—expression or aspiration—may be employed. The former method is the simpler and the one most frequently used. The patient is requested to take a deep inspiration, and then by the contraction of his abdominal muscles to expel the gastric contents upwards through the sound into the vessel prepared for their reception. The index finger should then be placed on the opening of the glass tube and the sound rapidly withdrawn. The closure of the lumen of the tube by the finger prevents the escape of the fluid contained within the sound and rubber tube. By removing the finger the contents are then allowed to flow into the receiving vessel.

For the aspiration method, Boas' aspirator, or other similar instrument, may be employed. A description of the different forms in use is unnecessary, since the expression method has the advantage of greater simplicity, and is quite as efficient.

(2) *Clinical examination of the gastric contents.*—*A.* Macroscopic examination.—The chief points to note in the macroscopic examination are the quantity, consistence, odour, and colour of the contents, as well as the presence of any foreign constituents, such as bile, blood, abnormal quantities of mucus, etc.

Before forming an opinion with regard to the *quantity,* one must feel sure that the total contents have at least been approximately obtained. The quantity is, of course, largely dependent on the form of meal taken. No conclusion can be drawn from the quantity of the contents as to the peptic activity of the gastric juice. The digestive activity of the juice can be better ascertained from the other macroscopic appearances of the contents. From the quantity found, however, one can infer whether motor insufficiency of the stomach is present or not. In cases of increased motility the stomach may be found nearly empty after Riegel's test meal within a shorter interval than the normal one of seven hours. In other cases the quantity is abnormally large, or the stomach is found to still contain food residues after the lapse of seven hours. These are primarily cases of motor insufficiency. As will be seen later, motor and secretory insufficiency run by no means always parallel.

Lastly, in some cases a larger quantity is found than that which was introduced into the stomach. These cases were hard to explain according to the former view that water was absorbed from the stomach. More recently, however, von Mering has shown that not only is little or no water absorbed by the stomach; but, on the contrary, the absorption of certain substances *e.g.* sugar, dextrin, peptones, and alcohol is associated with a secretion of water into the stomach.

It is further important to note the presence or absence of undigested food residues, and also whether these food residues are chiefly carbohydrate or chiefly proteid in nature. A fine uniform liquified mass suggests active peptonising power, accompanied, it may be, by hyperacidity. Undigested particles of food suggest the presence of subacidity. Abundant starchy residues associated with good proteid digestion justify the conclusion that hyperacidity is present. A certain opinion can of course only be formed with regard to the secretion after a detailed chemical examination. The necessity, however, of a careful preliminary macroscopic examination may be illustrated by the following examples:—In one case there may be found only a small quantity of hydrochloric acid, and yet the macroscopic examination may indicate a fairly complete peptonisation of the food. In another apparently similar case of subacidity, undigested food particles may be found in considerable quantity. If in these two cases an estimation of hydrochloric acid had alone been made, little or no difference in the conditions would have been detected. The consideration of the results obtained by macroscopic examination, combined with that of the results of the chemical examination, shows the important difference that in the former case the quantity of pepsin is much larger than in the latter.

The *odour* of the gastric contents is also of considerable significance. Normally they have no distinctive odour. In certain pathological conditions they have a somewhat rancid smell owing to the presence of free fatty acids. In cases of subacidity they sometimes have a putrefactive odour owing to the decomposition of proteids brought about by the growth of micro-organisms. In cases of intestinal obstruction or of an abnormal communication between the stomach and the intestines they may have a fæcal odour.

Mucus in considerable quantities, blood, bile, and pus are abnormal constituents that may occasionally be found.

Mucus, if present in large amount, can easily be recognised by its physical characters, and by the fact that it yields a precipitate on the addition of a little acetic acid. It is usually found in the upper part of the gastric contents when they have been collected in a vessel. The quantity of mucus normally present is small.

The Colour.—Blood, when present, may produce either a reddish or brown tint in the gastric contents. In the former case it has been freshly shed, and microscopical examination frequently reveals intact red blood-corpuscles. In the latter the hæmoglobin has become decomposed into methæmoglobin and acid hæmatin (see "Hæmatemesis").

Bile and intestinal juice are not infrequently met with even under normal conditions, the bile giving a greenish tint to the gastric contents. The usual tests for its chief constituents may be employed in its recognition. The presence of intestinal juice is proved by testing for the pancreatic enzymes.

Pus is very rarely found. It may be recognised on microscopical examination, and by means of the chemical tests used for its recognition in the urine.

In gaseous fermentation of the gastric contents the macroscopic appearances are very characteristic. The gastric contents, when collected in a vessel, show at least three distinct layers. The uppermost layer consists of foam formed by gas bubbles which rise to the surface, the middle layer is a more or less turbid fluid, and the lowest one consists of a sediment composed of fine carbohydrate residues. This condition is found under circumstances which allow stagnation of the gastric contents, as for example in motor insufficiency. The quantity of free hydrochloric acid is usually increased rather than diminished. In this respect gaseous fermentation is in striking contrast with other forms of fermentation, *e.g.* lactic acid fermentation, which are practically never found except in cases of subacidity. The mixture of gases present is occasionally inflammable. Chemical examination has shown the presence of variable quantities of the following gases:—Carbon dioxide, hydrogen, marsh gas, and acetylene, in addition to nitrogen and small quantities of oxygen. The last two gases are derived from atmospheric air which has been swallowed. Sulphuretted hydrogen is also occasionally found in the gastric contents, but it is not usually found in typical cases of gaseous fermentation.

For the examination of vomited material the same methods, macroscopic and chemical, are used as for the examination of the gastric contents obtained by the use of the sound. The vomited material may consist of mucus alone, mucus mixed with bile, or more or less digested food material with admixture of any of the pathological constituents already mentioned.

In certain cases the examination of the gastric contents after a test meal requires to be supplemented by a similar examination in a state of hunger *i.e.* in the morning before breakfast. The presence of any quantity above 50 cc. is to be regarded as pathological. Quantities above 50 cc. are usually found in cases of motor insufficiency or obstruction, and secondly in cases of hypersecretion.

B. The chief *morphological constituents* that may be noted on *microscopical examination* are the following:—Undigested food residues *e.g.* muscle fibres, starch granules, plant cells, fat globules, etc.; cell nuclei, squamous epithelium, columnar epithelium, mucous cells, blood and pus corpuscles, small portions of the gastric mucosa, particles of tumours, and various micro-organisms *e.g.* yeast cells, sarcina ventriculi, and numerous forms of bacteria.

C. Chemical examination of the gastric contents.—Prior to the chemical examination the gastric contents should be filtered. The filtrate alone is used for the examination.

1. The reaction of the filtrate should be ascertained by the use of blue and red litmus papers.

2. If the reaction be acid, the presence or absence of free hydrochloric acid should be ascertained. There are a number of organic colouring matters, which change in colour in the presence of free hydrochloric acid even when the acid is in very dilute solution; while they show no change of colour in presence of such strengths of organic acid solutions as are ever found in the gastric juice. The most commonly employed colouring matters are tropæolin 00 (saturated alcoholic solution), which is changed from yellow to red by free hydrochloric acid, methyl violet (·1 per cent aqueous solution), which becomes blue, and congo red, which is converted into a dark blue. The most important objection to the use of all these is that albumoses and peptone when present prevent or lessen the colour change by entering into loose combination with more or less of the hydrochloric acid. This objection does not hold good for Günzburg's test. Günzburg's reagent has the following composition:—Phloroglucin 2 gms., vanillin 1 gm., absolute alcohol 30 cc. A few drops of this yellowish fluid are mixed in a porcelain basin with a very small quantity of the filtrate, and the mixture evaporated to dryness over the open flame. If free hydrochloric acid be present, a carmine red residue is left; but if free organic acids only be present, a slight yellowish residue is left.

The simplest test for lactic acid is Uffelmann's reagent (2 per cent aqueous phenol solution to which is added a few drops of tincture of the perchloride of iron until the fluid becomes of a deep amethyst blue colour). This reagent should always be freshly prepared before use. It is not advisable to test for lactic acid by its means in the filtrate itself, as other substances, which share with lactic acid the power of converting the blue into a canary yellow solution, may be present in the filtrate. Amongst these may be mentioned glucose, phosphates, alcohol, oxalic, citric, and tartaric acids. About 10 cc. of the filtrate are concentrated to a thin syrup by evaporation on the water bath, thoroughly extracted with ether, using about 50 cc. in all. The ethereal layer is then separated, and evaporated to dryness on the water bath. The residue is dissolved in water, and tested by means of Uffelmann's reagent. For a quantitative estimation of lactic acid the extraction with ether must be more thorough. At least six extractions, using 100 cc. of ether each time, should be employed. The residue after evaporation of the ether should be dissolved in a known quantity of water and titrated with decinormal caustic soda solution. Each cc. of the decinormal alkali is equivalent to ·0090 gms. of lactic acid.

Butyric and acetic acids are also sometimes found in very small quantity.

The filtrate of the gastric contents may also be tested for the different forms of albumoses and for peptone, but as yet the examination has not obtained much practical importance.

A full account of the methods employed for these rarer and less important examinations will be found in the works referred to under "Literature."

The presence of pepsin may be recognised by means of an artificial digestion. 10 cc. of the filtrate are placed in a test-tube, hydrochloric acid being added, if necessary, until congo red paper is just turned blue, and a circular disc of hard boiled white of egg 1·5 mm. thick and 10 mm. in diameter is placed in the fluid. The disc may be cut from a hard boiled egg by means of a Valentine's double-bladed knife and a cork-borer. The test-tube is then placed on a water bath at 40° C. If the quantity of pepsin be normal, the albumin disc should be completely dissolved in a half or at most a whole hour.

To test for the presence of rennin, 10 cc. of the gastric filtrate are accurately neutralised by the addition of decinormal caustic soda solution. A few drops of the neutral fluid are then added to 10 cc. of milk in a test-tube, and the mixture is placed in a water bath at 40° C. If rennin be present in sufficient quantity, coagulation of the milk should be complete in ten minutes, or at most half an hour.

Quantitative estimations of the chief constituents of the gastric filtrate.

The *total acidity* may be estimated by the following method :—To 10 cc. of the gastric filtrate, diluted with a little water, a few drops of alcoholic solution of phenolphthalëin are added. Decinormal caustic soda solution is then gradually added to the mixture placed in a beaker until a permanent pink colour just forms. It is customary to express the degree of acidity so found by a figure stating the number of cc. of decinormal caustic soda necessary to neutralise the total acid in 100 cc. of the filtered gastric contents. If, for example, 5·7 cc. of decinormal caustic soda were required to neutralise the acid in 10 cc. of the gastric filtrate, then 57 cc. would be required to neutralise the acid in 100 cc., and the acidity would be 57 per cent.

Other indicators, *e.g.* litmus, may be used in the place of phenolphthalëin. The indicator chosen should be employed for all the estimations of one series. This is a necessary precaution, as the results for the acidity obtained by the use of different indicators do not always coincide when proteids are present in the acid fluid. If, for example, a drop of phenolphthalëin solution be added to a proteid one, which reacts neutral towards litmus, the addition of a small quantity of decinormal caustic soda will be found necessary before a permanent pink colour of the solution is secured. The total acidity varies within wide limits even under physiological conditions. 50-75 per cent may be given as the average.

The total acidity of the gastric filtrate is made up of the following factors :—(1) free hydrochloric acid, (2) combined hydrochloric acid, (3) acid salts, and (4) organic acids. The quantity of organic acids present is usually so small that it may be neglected. A method for the estimation of the most important one of these, namely, lactic acid, has already been given. For methods of estimating the other organic acids see "Literature."

Probably the simplest, and yet accurate method of estimating the free hydrochloric acid, is Fleiner's modification of that of Mintz. To 10 cc. of the gastric filtrate placed in a beaker are added about twenty-five drops of Günzburg's reagent. A drop of the mixture is cautiously evaporated to dryness over the open flame. A carmine red residue indicates the presence of free hydrochloric acid. For the quantitative estimation of the free mineral acid decinormal caustic soda is gradually added from a burette until a drop or two of the mixture yields no red residue on heating. After making a preliminary estimation a second one should always be performed, using the result of the first as a guide. The number of cc. of decinormal caustic soda used indicates the quantity of free hydrochloric acid expressed as so many cc. of a decinormal hydrochloric acid solution. The percentage of hydrochloric acid is obtained by multiplying this result by ·00365. Although this is always given as a method for the estimation of the *free* HCl alone, it is doubtful whether, after all, some at least of the combined HCl is not also estimated.

The *acidity due to acid salts* may be estimated by Leo's method. 10 cc. of the gastric filtrate are mixed with 5 cc. of a saturated solution of calcium chloride, and the fluid titrated with decinormal caustic soda in precisely the same manner as already described under estimation of the total acidity.

To 15 cc. of the gastric filtrate is then added 1 gm. of finely powdered pure calcium carbonate. The mixture is thoroughly shaken and then filtered through a previously dry filter into a well-dried beaker. Calcium chloride, acid phosphate, the excess of calcium carbonate, and carbon dioxide are present in the filtrate. The carbon dioxide is removed by passing a stream of air through the fluid by means of a hand bellows connected with a fine glass tube dipping into the fluid, or by means of an aspirator. 10 cc. of the filtrate, now freed from carbon dioxide, are accurately measured out, mixed with 5 cc. of a saturated solution of calcium chloride, and titrated with decinormal caustic soda as before. The latter result gives the acidity alone due to acid salts. The former result gives the total acidity.

A few words are necessary to explain the principles upon which Leo's method is based. Solutions of free acids on being shaken with calcium carbonate in the cold are completely neutralised, while fluids containing acid (*i.e.* dihydrogen) phosphates after similar treatment retain their acid reaction. In a mixture of free acids and acid phosphates, one can therefore exclude the acidity due to free acids by the addition of calcium carbonate, and then estimate in the filtrate the acidity due to acid phosphates. The soluble calcium salts which are formed by the interaction of calcium carbonate with the free acids present (chiefly hydrochloric) react with sodium dihydrogen phosphate to form sodium chloride and calcium dihydrogen phosphate. One then estimates the acidity of the latter salt, which requires twice as much caustic soda to convert it into the neutral calcium phosphate as is necessary for the neutralisation of the corresponding acid phosphates of sodium and potassium. The following equations will help to render the matter clear:—$2NaH_2PO_4 + 2NaOH = 2Na_2HPO_4 + H_2O$; whereas $2\ NaH_2PO_4 + 4\ NaOH + 3CaCl_2 = Ca_3(PO_4)_2 + 6\ NaCl + 4H_2O$.

To overcome this difficulty, one of two expedients may be employed. The total acidity may be estimated in the usual way without the addition of calcium chloride. The second titration may be carried out exactly as before. The number of cc. of caustic soda used in the second titration would, however, require to be divided by two. This division by two becomes unnecessary if both estimations are performed under the same conditions, namely, the addition of excess of calcium chloride. Since the second method is the simpler, it is almost invariably employed.

The total quantity of hydrochloric acid—free and combined—is easily ascertained from the results already given. It is equivalent to the total acidity minus that due to acid phosphates and lactic acid if present.

For the estimation of the total "physiologically active" hydrochloric acid (free and combined) numerous other methods have been employed; of these Martius and Lüttke's appears to be the most accurate. As the method, however, is somewhat too complex for clinical use, a brief sketch of the principles upon which it is founded must here suffice. The total chlorides in 10 cc. of the gastric filtrate are first estimated as in the urine by Volhard's method. The quantity of chlorine in the ash of another 10 cc. is then estimated and reckoned in terms of hydrochloric acid. By subtracting the latter result from the former, one obtains the chlorine present as free and combined hydrochloric acid.

The loosely combined hydrochloric acid is equivalent to the total acidity minus that due to acid phosphates, free hydrochloric acid, and lactic acid if present. The combined hydrochloric acid is in loose union with amido-acids, albumoses, and peptones. The total hydrochloric acid is sometimes termed the "physiologically active" acid, because the loosely combined is as important in peptic digestion as the free.

THE MOTOR ACTIVITY OF THE STOMACH may be tested by several methods. 1. Probably the best of these for practical purposes is that of *Leube.* Seven hours after the taking of a test meal similar to that of Riegel, an examination is made with the gastric sound. Under normal conditions the stomach should then be found empty; but in cases of motor insufficiency the amount of fluid varies according to the severity of the case.

2. The *salol method of Ewald and Sievers* for testing the motor efficiency of the stomach is founded upon the fact that salol—a compound of salicylic acid with phenol—is not decomposed in acid solution, but readily undergoes decomposition in alkaline solution into phenol and a salicylate of the alkali employed. This decomposition cannot take place in the stomach; but occurs whenever the salol passes into the small intestine. The salicylate thus formed is absorbed, and excreted in the urine as salicyluric acid, which gives a violet colour with ferric chloride. If 1 gm. of salol be given to a healthy individual during digestion, the reaction with ferric chloride should appear in the urine after half or at most three-quarters of an hour. Delay in the appearance of the reaction indicates a

retention of the contents of the stomach. The salol test has, however, turned out to be far from reliable. Stein has shown that salol is absorbed from the stomach itself, and that the products of its decomposition may afterwards be detected in the urine.

3. A third method—that proposed by Klemperer—consists in introducing into the stomach, previously freed from food residues, 100 cc. of olive oil. After two hours the gastric contents are obtained by means of the sound, the complete removal of the oil being afterwards ensured by repeatedly washing out the stomach with water. The oil, after being separated from the water in a separating funnel, is measured. The diminution in quantity expresses the amount of oil which has been expelled into the intestines. Klemperer found that under normal conditions 70-80 cc. of oil were expelled into the intestines. This method, although of considerable scientific value, is obviously one ill-suited for practical use.

4. Lastly, a short reference to Einhorn's electrical method for the examination of the motor activity of the stomach must suffice. For this purpose he uses an ingeniously contrived instrument called the gastrograph. Although indicating all movements of the gastric contents, it is chiefly of use as a recorder of those gastric movements which complete the mechanical subdivision of the food and ensure its thorough admixture with the gastric juice. The method has not so far proved of much practical utility.

Pathological variations of gastric digestion may be divided into secretory, motor, and sensory.

The variations in secretory activity may be either in the direction of increased or of diminished activity, and may affect one or all of the constituents of the gastric juice. Whereas under normal conditions the gastric mucosa only secretes in consequence of some stimulus, in hypersecretion it continues to act during the intervals between meals, and, in consequence, the stomach is found to contain fluid even during hunger. This hypersecretion or gastro-succorrhœa may be intermittent or chronic, and is usually associated with more or less sensory disturbance.

Diminished secretion of gastric juice is found, especially in atrophic conditions of the gastric mucosa, *e.g.* in adenia ventriculi.

Anomalies of secretion affecting individual constituents of the gastric juice are usually limited to variations in the quantity of hydrochloric acid produced, the quantity of pepsin remaining fairly constant, and may manifest themselves, either as a subacidity or a hyperacidity of the gastric juice. The total acidity in such cases may reach values of from 100 to 150 per cent, while the acidity due to free hydrochloric acid may amount to 50 to 60 per cent. The alterations in acidity in the different pathological conditions affecting the stomach (carcinoma, gastric catarrh) are referred to under their special headings.

Diminished motor activity of the stomach may affect the muscular walls of the organ as a whole, *e.g.* in atony of the stomach, or be limited to certain parts, as in pyloric or cardiac insufficiency.

Increased motility may be due to increased resistance to the expulsion of the contents as in pyloric stricture, or it may be established to compensate for secretory insufficiency, or it may be due to irritation, as in hyperchlorhydria, or lastly it may be nervous in origin. It may be general, as in peristaltic unrest of the stomach and in vomiting, or it may take the form of spasmodic contractions localised to the cardiac or pyloric orifices.

While digestion is normally unaccompanied by sensation, in pathological conditions the varieties of sensory disturbance that may occur, are so numerous that reference must be made to the special articles on diseases of the stomach.

Bile

The secretion of bile takes place continuously, in this respect differing from the true digestive secretions. At the present time bile is regarded mainly as a fluid which contains certain effete products destined for excretion, some of these, however, being absorbed by the intestine after their discharge from the bile duct. Certain substances which are insoluble in such a watery solution as urine, can be dissolved and so excreted by the bile. The most important biliary constituents are formed in the liver, not being found in the blood either normally or after removal of the liver; but, if the outflow of bile be hindered, the biliary constituents appear in the blood and are partly deposited in different tissues, partly excreted by the kidneys, giving the urine a brownish-green colour.

Physiological Action.—Until comparatively recently it was assigned a very important rôle *in digestion;* but now it is only regarded as of importance in the absorption of fats. It does not exercise any direct deterrent action on putrefying processes so far as can be made out. The digestion of proteids and carbohydrates is practically unaffected by its withdrawal; but only about half the normal amount of fat is absorbed, the rest being excreted in the fæces. This withdrawal of fat in the fæces allows the action of putrefaction organisms on the proteids, because the latter become coated with fat, and so the action of the pancreatic juice is prevented, and decomposition ensues. So long, however, as excessive quantities of fat in the food are avoided, neither the general condition, *e.g.* nitrogen equilibrium, nor digestion suffers. It is exceedingly difficult to study the action of drugs on the bile secretion, because the flow is such a variable one even under ordinary conditions, and as a result of this the conclusions which different investigators have come to are very contradictory. It seems probable that no substance, with the exception of certain of the bile constituents themselves, acts as a true cholagogue. The pressure of the bile in the ducts can rise to a much greater height than that of the blood in the portal system. The temperature of the blood in the hepatic vein is higher than that in the portal, showing that the cells have been undergoing active metabolic changes; but of course this applies not only to the biliary, but also to the glycogenic function of the liver.

Mode of Origin of Constituents.—The only bile constituent of which we know more or less definitely the mode of origin is the bile pigment. This pigment occurs only in animals which contain hæmoglobin in their blood. It is certain that the blood pigment is either set free in the liver from the breaking down of red blood-corpuscles, or it is set free elsewhere and carried to the liver, where it is split up into an iron-holding radicle, hæmatin, and albumin. The former loses its iron and takes up water to form bilirubin, the iron being retained in the liver, perhaps helping to form hæmoglobin later.

A very small quantity of iron is excreted in organic form in the bile, not nearly sufficient to account for the amount present in the hæmoglobin before destruction. After injection of hæmoglobin into the blood, there is an increased production of bilirubin; but this does not result in an absorption and deposition of pigment until the amount becomes so great that the bile capillaries or smaller bile ducts become blocked. Any agents which can break down the red blood-corpuscles setting free hæmoglobin can produce bilirubinuria if the amount of disintegration be great enough to produce obstruction in the bile capillaries. On obstruction to the bile outflow, the resultant absorption takes place in the larger bile ducts, through the lymphatics into the thoracic duct. Bilirubin can be formed from hæmoglobin outside the liver, as, *e.g.* in old blood extravasations, hæmorrhagic infarcts, etc. The pigment which crystallises out under such conditions was first of all described by Virchow as hæmatoidin, but it is now known to be identical with bilirubin. There are many other facts which bring out strongly the intimate connection between the blood and bile pigments. Only one of these need be referred to, namely, the formation and excretion in the urine of the important iron-free blood pigment, hæmatoporphyrin, in certain conditions, *e.g.* after sulphonal poisoning (see "Hæmatoporphyrin"). This pigment is isomeric with bilirubin. The chief pigment in human bile is not, however, bilirubin but biliverdin, which gives the green colour to the fresh biliary secretion. It is rapidly reduced in the intestine, from the action of bacteria, into hydrobilirubin and derivatives of that pigment. The source of the other constituents of the bile has not been so well made out. *Cholesterin*, a constituent probably of every living animal cell, is, like the bile pigment, an effete product. It is derived from the metabolism of the cell protoplasm, and is distinguished by the resistance which it offers to further decomposition, and also by its tendency to form gall stones. With regard to the *salts of the bile acids*, glycocholic and taurocholic, they are the only bodies which can undoubtedly act as cholagogues, undergoing absorption by the intestinal mucous membrane after their excretion into the duodenum. There can be no doubt, however, that this absorption takes place, not in the duodenum, but in the jejunum and ileum. As these acids undergo partial absorption, alterations in the amount of proteid in the food do not affect to any extent their excretion in the bile. Thus, after increasing the amount of proteid in the food, the salts of the bile acids are barely increased at all in the bile. Their absorption may take place directly into the blood-vessels of the intestinal mucosa, although probably the lymphatics form the main channel. These salts probably play an important part in aiding the absorption of other substances by the intestine, for example, soaps of the alkaline earths and fats. The other constituents of the bile are not of so much importance. The body, which lends the peculiar stringy consistence to the bile, namely, *mucin*, is secreted by the gall bladder and larger bile ducts. In man it is a true mucin; but in the ox it is supposed to be a nucleo-albumin. There are also present in the bile small quantities of *fat*, *lecithin*, and *soaps*.

The *amount* secreted can only be estimated in cases of biliary fistula, and such estimations are not reliable, because a certain amount of the bile is normally absorbed by the intestine, and acts upon the biliary function of the liver. If it be then withdrawn by means of a fistula, this natural cholagogue is removed, and so less is excreted than what would normally be the case. The quantity also varies with the amount of water taken and the nature of the diet, animal food increasing it to the most marked extent. The maximum of flow occurs some hours after food has been taken; the larger the meal, the longer the interval before the maximum appears. Exceedingly little is known as to the action of the nervous system on bile secretion. There seem to be certainly no true secretory nerves, but the secretion may be affected by vasomotor influences. If the blood of the portal vein be allowed to pass directly into the inferior cava without passing through the liver, the secretion of bile still goes on, and the same holds good after obstruction of the hepatic artery. After splanchnic stimulation there is a diminished flow, with contraction of portal vessels, while after section the opposite occurs. The expulsive action of the gall bladder and larger bile ducts is under the influence of the nervous system, peristaltic waves of contraction, similar to the intestinal ones, occurring along their smooth muscular fibres. These may also be stimulated reflexly from the gastric mucous membrane. The removal of bile from the liver through the ducts is caused by the back pressure of the newly formed secretion, and also from the pressure on the liver during inspiration.

PATHOLOGICAL ALTERATIONS.—It may undergo *pathological alterations*, of which the chief are due to obstruction to the bile outflow when the pigments and salts of the bile acids are absorbed, and the fluid left in the dilated channels is often found to contain only traces of the true constituents with an excess of mucin (see "Jaundice").

Under certain conditions, the nature of which we do not yet know, *gall stones* may form in the ducts. These vary in size, form, and chemical composition. There are two important forms met with in man, cholesterin and pigment chalk stones, the former being by far the more frequent. They are white or faintly yellow in colour, and are specially characterised by their low specific gravity, which is less than that of water. Absolutely pure cholesterin stones are rare, there being usually a bilirubin chalk nucleus around which the cholesterin has crystallised out. The pigment stones are much more easily broken down by pressure than the others, and are usually much smaller and heavier. They very often contain traces of copper and a larger or smaller quantity of other bile pigments in addition to bilirubin, *e.g.* biliverdin. When numbers of gall stones have collected in the ducts, they present a facetted appearance, owing to the pressure to which they have been subjected. Very often small stones or gravel, composed of calcium carbonate tinted with bilirubin, are passed. The occurrence of traces of heavy metals, *e.g.* Ca, Zn, As, emphasises the importance of the bile as a medium for excretion. With regard to the causes leading to the formation of gall stones, it has been surmised that in the case of cholesterin ones, two factors may have played a part, viz. insufficiency of bile salts to keep the cholesterin in solution, or excessive formation of the latter. The most probable cause leading to the formation of both kinds of calculi is obstruction to the bile outflow owing to catarrhal conditions of the ducts, when the excessive secretion of mucus or the shedding of epithelium may at least aid in their production.

PANCREATIC SECRETION.—NATURE AND METHOD OF SECRETION.—The most important changes which the food stuffs undergo are the result of the action of the *pancreatic juice.* The acid chyme is gradually neutralised by the clear alkaline fluid which is secreted by the cells lining the pancreatic acini, and which is poured into the duodenum through Wirsung's duct. If the fluid be obtained from a fistula, it is found at the outset to have a specific gravity of about 1·03; but, after some time has elapsed, it becomes poorer in proteids and of lower specific gravity. In carnivora and omnivora the secretion is not a continuous one. During activity the cells of the pancreatic acini undergo distinct alterations. Thus at the beginning of digestion the granules of zymogen gradually disappear from the inner zone until the whole cell at last appears clear. Then the granules are again formed in the inner part, the outer zone acting as the storehouse for the material out of which the zymogen is formed. These zymogen granules are only transformed into the active ferment after the gland has been exposed to the air for some time, or after it has been subjected to the action of weak alkalies or acids. The *amount* of the juice secreted in twenty-four hours is impossible to estimate accurately, as in cases of fistula it undergoes marked alterations, rapidly also becoming poorer in solids. As in other similar secretory processes, the secretion is accompanied with vascular dilatation.

NERVOUS MECHANISM.—If the vagus be stimulated, there is an increase in the amount secreted, the fluid also being thick in consistence; while, if this nerve be cut, and its central end stimulated, the flow is greatly diminished, or may be stopped altogether. A rather important cause of stoppage of secretion is vomiting, here also the influence of the vagus making itself felt. It is an interesting fact that the acid chyme, on coming into contact with the duodenal mucous membrane, causes an increased pancreatic secretion, and this brings about neutralisation, while alkalies have the opposite effect. This reflex secretion persists even after the vagi and sympathetic have been cut, its occurrence being then probably due to the presence of a local centre perhaps in the neighbourhood of the pylorus. The maximum flow occurs about three hours after food has been taken, then sinks for about the following four hours, when it may again rise. The greater the flow, the poorer is the juice in solids.

ACTION OF THE ENZYMES.—*The ferments in the pancreatic juice* are four in number, the most important one being *trypsin*, which acts upon proteids, transforming them into more soluble bodies.

Unlike pepsin, it acts best in a weakly alkaline solution, although it is also active in neutral or faintly acid ones. Still even with weak organic acids, although at the outset the action is almost as marked as in slightly alkaline media, the ferment is slowly destroyed, disappearing, however, much more rapidly with weak inorganic acid solutions. Thus it is useless giving such preparations as pancreatin by mouth, because trypsin is rapidly destroyed by the acid gastric juice. The first action which it exercises on proteids is simply a solvent one, and it is doubtful whether any alkali-albumin is found or not. The digestion passes so rapidly through the stage of primary albumoses that it is often difficult to detect their presence, but deutero-albumose (a secondary one) is formed in large quantity, and then peptone makes its appearance. But the most characteristic feature, from the chemical standpoint at least, in tryptic digestion is the further decomposition of the peptone. Until recently it was believed that all the peptone did not undergo decomposition, but that a part was resistant, this being termed antipeptone, while the rest, which was split up into amido-acids (such as leucin, tyrosin, and aspartic acid), ammonia, etc., was termed hemipeptone. Siegfried regarded antipeptone as identical with an acid which he found in paired combination in muscle and milk, namely, carnic acid; but it seems unlikely that this is the case, as Kossel, Kutscher, and others have shown that Kühne's antipeptone is really a mixture containing certain bases, called the hexone bases (lysin, histidin, and arginin). These bodies are only formed after very prolonged digestion. The point of importance is that, in all probability, if only digestion be continued long enough, all the peptone can be broken down. Now, although this is true for artificial tryptic digestion, the action is a much more limited one in the intestine, very little leucin and tyrosin ever being formed under normal conditions. In fact, it is probable that the pancreatic juice is mainly required to render the proteids soluble and ready for absorption in cases where such large quantities have been taken that, if this action did not take place, a great loss of proteid would result, this material undergoing decomposition in the lower parts of the intestinal canal. As we shall describe more fully later, one is compelled to admit that even the transformation into albumoses and peptones is not absolutely essential because the forerunners of these (soluble albumins and albuminates) are themselves capable of absorption by the intestinal mucosa.

The products of a pancreatic digestion easily undergo putrefaction, and hence in artificial digestive fluids such an agent as thymol or chloroform requires to be added. In addition to the substances mentioned above, a chromogen termed tryptophan, the nature of which has not yet been fully made out, is formed about the time that the amido-acids appear. A small amount of free ammonia also appears during digestion. Such is very shortly the nature of the hydrolytic decomposition of a simple proteid under tryptic digestion. Complex proteids are also more energetically attacked by trypsin than pepsin. Thus nucleo-albumins have not only their albuminous constituent easily broken down, but the resistant nuclein radicle is also slowly dissolved.

There are in addition three other enzymes which occur in the pancreatic juice, an amylolytic, a fat-splitting, and a milk-curdling one. The *amylolytic* ferment, so far as we at present know, acts on the polysaccharides in the same way as ptyalin. Thus starch and glycogen undergo hydration and are split up into the dextrins, which were described under salivary digestion, and maltose. The amount of the final product, maltose, depends on the relative quantities of enzyme and polysaccharide. The monosaccharides undergo no alteration, but are absorbed without alteration, while cellulose is not affected by the juice.

The action of the *pancreatic juice on fats* is a more complex one. The acid chyme is neutralised by the bile, pancreatic juice, and the secretions from the intestinal glands, and then, if fatty acids be present, emulsification of fats can at once take place; but the fat-splitting ferment assists in this action, setting free fatty acids from the neutral fats. This fat-splitting action does not require to be a very marked one, because a very small quantity of free fatty acid is able, in the form of a soap, to emulsify a large quantity of fat. Later on, the mode of absorption of the emulsion will be referred to. Lecithin is also split up by this ferment. Bile seems to assist this ferment in its action. Like all other enzymes, it is easily destroyed by boiling, and the same occurs if the gland be exposed to the air for a short time, as in the artificial preparation of pancreatic extracts.

The milk-curdling ferment of the juice seems to be identical with rennin, only its mode of action has not been so carefully worked out as that of the ferment derived from the stomach.

PATHOLOGICAL ALTERATIONS in the juice have not been much investigated. In certain inflammatory conditions affecting the pancreas the juice secreted is thin and inactive, and in other pathological conditions there may be a stoppage of the flow, due to the ducts being blocked up. The pancreas, however, plays an important part in the metabolism of glucose, which may be referred to here. If the gland be removed entirely from dogs, diabetes of a severe type at once sets in, and death ensues in a short time; but if a small portion be left, so long as this does not undergo atrophy, only traces of glucose appear in the urine. As soon as the remaining piece atrophies, marked glycosuria sets in, and within a few weeks or months the animal dies. In such cases glucose is excreted even when no carbohydrate is given in the food, and if any be given by the mouth, it is wholly excreted in the urine. The blood in such cases shows a high percentage of glucose—0·3-0·5 per cent. It is important to note that mere withdrawal of the juice from the intestine does not produce diabetes, nor does ligature of the pancreatic vein. At present we do not know in what way the pancreas exercises an influence on the metabolism of glucose. In a few cases of diabetes the pancreas has, on post-mortem examination, been found to be atrophied, or otherwise pathologically altered (see "Diabetes").

INTESTINAL SECRETION.—The succus entericus is secreted by Lieberkühn's glands of the small and large intestines. It is obtained usually from fistulæ (Thiry-Vella), when a piece of small intestine, about 50 cm. long, has been resected with the mesentery attached and the upper and lower openings sewed into the abdominal wall.

It is a clear yellow fluid of markedly alkaline reaction, containing usually a fairly large quantity of solids, although the amount of these varies very much. With regard to the *enzymes* present there is a great deal of contradictory evidence. It seems most likely that it contains no ferment which acts on proteids or fat; but it certainly contains an inverting ferment, and very probably more than one. In some animals the secretion appears to be a continuous one, in others it only occurs after stimulation of the mucous membrane. Experimentally one may use either mechanical, chemical, or electrical stimuli. Usually the amount secreted begins to increase about one hour after food, and gradually rises to about the third hour, when it falls again. Very little is known as to *nervous influences* exerted on this secretion. If all the nerves, passing to such an intestinal loop as has been described above, be cut, fluid accumulates in the sac. Stimulation or section of the vagi seems to have no effect. With regard to the action of the juice on the food, there seems always to be a slight diastatic action, starch being converted into maltose; but more important is the inverting action transforming maltose into glucose and cane-sugar into glucose and levulose. Lactose does not seem to be inverted by the juice, although it may be in its passage through the intestinal mucosa. The inverting action takes place in the presence of antiseptics, but is destroyed by boiling.

In the lower part of the small intestine, where putrefactive organisms possess a suitable slightly alkaline medium, the proteids undergo changes which are at the outset similar to those produced by the gastric and pancreatic secretions. That is to say, insoluble albumins are transformed slowly into soluble forms (albumoses and peptones), and in all probability this action is due to enzymes secreted by the bacteria. These organisms do not attack unaltered albumin in the upper part of the intestine, because there they have a supply of albumoses and peptones ready for assimilation. Recently an elaborate series of experiments has been carried out in order to discover whether micro-organisms are necessary in intestinal digestion, and it has been shown that guinea-pigs which had been born in a sterile medium could live there even when their food was absolutely free from organisms. This of course does not touch the question as to whether bacteria may not be of use in aiding the action of the intestinal enzymes. And again there are substances present in the usual diet which are unaffected by the other enzymes, but are dissolved by bacterial action, *e.g.* celluloses. These micro-organisms can also break down proteids along different lines from the proteolytic enzymes. In the first place the aromatic radicle may undergo a variety of decompositions. Thus tyrosin is not only formed itself, but from it other benzol derivatives are obtained by reduction or oxidation, ending with the production of phenol, and in addition there are produced aromatic substances of a different order which are not found in ordinary tryptic digestion, namely, indol, skatol, and derivatives of these. In the second place, bodies of the fatty series are formed often in fairly large quantity, *e.g.* leucin and volatile fatty acids such as butyric, valerianic, and caproic acids. There is no free oxygen in the intestine, owing to the continual formation of hydrogen and sulphuretted hydrogen from bacterial action, so that any parasites which are present must get it indirectly from the capillary blood of the mucosa by keeping as close as possible to the intestinal wall. The amount of oxygen which they require is very small.

Ptomaines.—There are also bodies of a basic nature derived from the decomposition of proteids which normally are not formed in the intestine during life These bodies, termed ptomaines, do not appear until about three days after death. They are formed also when the intestinal bacteria are grown in different culture media. As a rule these bodies, which belong usually to the fatty series, are not very poisonous if they be produced rapidly after death; but those which make their appearance later are often extremely poisonous even in minute doses. The best examples of the non-poisonous class are *cadaverin and putrescin*, while neurin, derived from the decomposition of lecithin, is the best example of the poisonous series. This body is formed, unlike the ordinary poisonous ptomaines, comparatively rapidly after death, usually about the third day. One must bear in mind the possibility of the formation of such bodies in tinned meats and decomposing flesh of any kind, and also that similar bodies may be formed even during life in certain animals, *e.g.* mussels (see "Diet"). It is a comparatively rare occurrence to meet with the formation of those bodies under pathological conditions in the organism, but they do occur in the fæces in cholera, especially putrescin and cadaverin. The primary poisonous products of bacteria are usually of the nature of globulins or albumoses.

Not only are proteids acted upon by micro-organisms in the lower parts of the intestinal canal, but fats are split up into glycerine and fatty acids, and carbohydrates undergo manifold fermentations or are simply hydrated. So long as there are carbohydrates present in the small intestine which have not undergone fermentation, the proteids are protected from the action of micro-organisms. Thus a milk diet, from the presence of lactose, prevents decomposition of proteids owing to the formation of lactic acid. The influence which unabsorbed fats exercise on the putrefaction of proteids has already been referred to. In the comparatively short small intestine of carnivora, and even in that of omnivora, very little putrefaction is set up compared to that occurring in the large intestine. Putrefaction in both small and large intestines is limited, however, to a comparatively small amount of the food stuffs present, because, in the first place, micro-organisms, by rendering these substances more soluble, transform them into material which is more easily absorbed, and when they do break down proteids further, they form small quantities of substances, such as phenol, which check putrefaction; while in the last place, owing to the great absorption of water in the large intestine, the conditions under which the bacteria grow are rendered less suitable. Before taking up the subject of intestinal movements, etc., it is necessary to refer shortly to the modes of absorption of the digested material, and also to the nature of its transmission through the organism.

ABSORPTION.—The part which the gastric mucous membrane plays in absorption is still doubtful. Although very little water is absorbed, a fairly large quantity of soluble salts, albumose, sugar, alcohol, etc., is taken up;

but the main channel of absorption is through the intestinal mucosa. The amount of a food-stuff absorbed is independent of its osmotic equivalent.

ABSORPTION AND METABOLISM OF PROTEIDS.—Although the absorption of proteids is rendered an easier and more rapid process by their peptonisation, there can be no doubt that soluble albuminous substances, such as syntonin and other albuminates, can be absorbed without undergoing further alteration. Modern treatment by rectal feeding is based on this fact, for in the large intestine the transformation of proteids into albumoses and peptones takes place only to a slight extent, not nearly sufficient to account for the fact that patients can by this means be kept in nitrogen equilibrium. This is true not only for the absorption, but also for the assimilation of soluble albuminous substances. Thus one can inject the blood-serum of another animal into the veins of a dog without albumin afterwards appearing in the urine. The formation of large quantities of albumose or peptone seems in fact to be deleterious, as the intestinal mucosa is apt to suffer from excessive stimulation. When albumoses or peptones are absorbed, they are not carried as such to the tissues, but undergo polymerisation into albumins in the intestinal mucosa.

Neither albumoses nor peptones are present in the blood at any time during digestion, nor are they stored up in the intestinal wall, and yet if they be allowed to lie in a separated intestinal loop, they rapidly disappear. They must then undergo some alteration in the intestinal wall, and if the blood in the mesenteric or portal vein be examined after a meal rich in proteids, it is found to be much richer in albumins than before digestion, while the lymph in the thoracic duct shows no alteration in its percentage of proteid. Ligature of the thoracic duct does not prevent the absorption of proteid, nor the subsequent rise in the excretion of nitrogen in the urine. These facts go to prove that the absorbed albumoses are transformed into albumins, and that the channels into which the latter pass are the blood and not the lymph vessels. The means by which this transformation has been brought about are unknown. The leucocytes certainly do not seem to take up the albumose and peptone and transform them into albumin, because no evidence has been brought forward that they are capable of effecting such a transformation. The great mass of absorbed proteid seems to act as a source of energy without being taken up by the tissues and organised, although a small amount must supply the place of the cell proteids, which are always undergoing degeneration. The amount of nitrogen excreted during the day in the healthy adult is equal to the amount absorbed in that period—that is to say, the organism breaks down the same amount of proteid that it absorbs, a condition referred to as *nitrogen equilibrium.* The small amount of nitrogenous waste products which the tissue proteids furnish to the blood when they break down is balanced by the amount of proteid taken up from the blood (circulating proteid) for the regeneration of the tissues. It is exceedingly difficult—one may say impossible—at present to follow out the changes which the proteids undergo in their decomposition. The end products of their metabolism are well known, but the first katabolic changes which they undergo in the cell protoplasm we can only surmise. *Urea* is the principal nitrogenous end-product in mammals, while in birds uric acid takes its place. In both cases ammonia salts play an important part as forerunners of these end-products. When blood containing ammonium salts of formic, acetic, or lactic acid circulates through a fresh liver, urea is formed in appreciable quantity. Here, in all probability by a process of oxidation and synthesis, ammonium carbonate is formed, then, by loss of water, ammonium carbamate, and finally urea. It is probable that one of the most important non-nitrogenous decomposition products of proteid metabolism is sarcolactic acid, which becomes distinctly increased in the blood after proteids have been taken in the food, and falls correspondingly in conditions of hunger. This acid seems to be formed in most organs, *e.g.* lungs, kidneys, etc., when blood is transfused through them, and its percentage in muscle rises during exercise. Its ammonium salt is transformed into urea in the liver. If oxidation processes be hindered in any way, there is an increased formation and excretion of this acid. There are many other derivatives of proteid metabolism, *e.g.* glycocoll, acetone, etc., which may appear in the urine as such or in combination with other substances (see "Urine"). The sulphur and phosphorus present in certain proteids are in large part oxidised to their full extent and excreted as simple or paired sulphates and as phosphates (see "Urine").

ABSORPTION AND METABOLISM OF CARBOHYDRATES.—Under ordinary conditions the glucose is only taken up by the blood capillaries, but if there be a great excess of sugar in the intestine, some may also pass into the lacteals. Even after a diet comparatively rich in carbohydrates, the amount of glucose in the lymph flowing from the thoracic duct shows no increase, while the blood in the portal vein shows a rise from the normal, 0·11 per cent, to about 0·4 per cent. In order to prevent the loss of such an easily diffusible substance, the liver acts as a storehouse for the glucose, transforming it first of all into a less soluble form, glycogen, and giving it out again to the blood in the form of glucose when the percentage of the latter in the blood tends to fall before normal. By excluding carbohydrates from the diet one can gradually lower the amount of glycogen in the liver; while if one inject glucose into the blood of an animal whose liver has been rendered free from glycogen, the percentage of the latter in the liver can be gradually raised. It seems probable that these transformations of glucose into glycogen and glycogen into glucose are the results of the activity of the liver cells, and not of the action of an enzyme produced by the latter. The muscles and many other tissues can effect this dehydration and polymerisation of glucose into glycogen as well as the liver. In fact, glycogen seems to be present at least in every living cell at some stage or other in its life history. The muscles seem to store glycogen before the liver does, and they certainly retain it longer. Thus it is possible to exhaust the liver while the muscles still contain a fairly large quantity. In frogs the muscles can also store up glycogen even when the liver has been removed. One may say that approximately 150 grammes of the sugar that has been absorbed by the intestine are stored up as glycogen in the liver, and a slightly larger quantity in the muscles and other tissues of the body. When a larger quantity of carbohydrate is absorbed, then the liver and muscles would in all probability be unable to convert the excess into glycogen, and the percentage of glucose in the blood might rise to such a height (over 0·3 per cent) that the kidneys could no longer retain it, and the condition of glycosuria would result. But it is probable that there is another reason for temporary alimentary glycosuria in healthy individuals. In cases where large quantities of carbohydrate have been taken with a free supply of liquids, absorption may take place by way of the lacteals, and the sugar may reach the general circulation without passing through the liver. The percentage in the blood in such a case may be so high that temporary glycosuria results. When very large quantities of disaccharides, such as cane or milk sugar, are taken, some may escape inversion in the intestine and be excreted unchanged in the urine. One must remember, however, that the bowels may act as a safety valve for the removal of the excess of sugar, diarrhœa being set up by the intestinal irritation. Proteids may also act as a source of glycogen, so that even when no carbohydrates are given in the food, a liver which has been rendered free from glycogen can store it up again if sufficient proteid be given in the food. It is important also to remember that the glycogen may accumulate in the liver from the action of substances which prevent the liver cells transforming it into glucose, *e.g.* glycerine, antipyrin, chloral, etc. Inversion of the disaccharides is necessary before they can be transformed into glycogen. The sugar circulating in the blood is normally oxidised by the tissues, furnishing finally carbonic acid and water, and in this action the muscles play an important part, as the glucose is the chief source of muscular energy. When this action of the tissues does not come into play, the percentage of glucose in the blood rises until the kidneys can no longer refrain from excreting it (see "Diabetes"). The influence of the nervous system on the glycogenic formation and transformation in the liver is probably a vasomotor one. Thus after puncture of the floor of the fourth ventricle, poisoning with nitrite of amyl, curari, etc., the resultant glycosuria is due to an increased transformation of glycogen into glucose. The influence of the pancreas on the metabolism of glucose has already been referred to.

Absorption and Metabolism of Fats.—This can take place to some extent even when no pancreatic juice or bile is allowed to act on the chyme; but both of these are of great importance in aiding absorption. In many cases the mixed contents of the small intestine are acid in reaction (as in dogs), and yet emulsification of the fats can take place if the pancreatic juice have free access. It is not necessary that there should be sufficient alkali present to neutralise all the fatty acids, because not only is a small amount of soluble soap able to emulsify a fairly large quantity of these acids, but in addition the salts of the bile acids can easily dissolve them and so aid absorption. Soaps and fatty acids pass partly into the blood capillaries, partly into the lymphatics, but they are transformed into fats during their passage through the intestinal mucosa, for after they have been given in the food, the lymph in the thoracic duct contains only fats.

There is no reason for believing at present that the absorption of fats takes place entirely, or even most largely, in the form of solutions of soaps, and not as an emulsion of soaps, free fatty acids, and fats. After absorption of fat, the cytoplasm of the columnar cells of the intestinal mucosa is found crowded with fat globules, while none are to be seen in the striated hem. This is no proof, however, that the fat has not passed through in the form of an emulsion, although it has been regarded as supporting the view that the fats are absorbed in the form of solutions of the soaps, and that the latter undergo in the columnar cells synthesis with glycerine to form fats. Undoubtedly the contraction of the smooth muscular strands in the villi aids at least the passage of the chyle through the lacteals. Fats of low melting-point are more rapidly and completely absorbed than those which melt at a higher temperature. In many cases when fats foreign to the animal experimented upon are absorbed, they are deposited unaltered in the tissues; but in man the absorbed fats are not necessarily deposited in the form in which they were taken in, being usually transformed into the ordinary mixed fats of the tissues. There can be no doubt that the synthesis of fatty acids and glycerine into fats can and does take place in the columnar cells of the intestinal mucosa; but as has already been said this does not exclude the other method of fat absorption—emulsification. Carbohydrates and proteids may also act as sources of fat, but the proof of this cannot be given in this article (see "Diet"). The fats are deposited in various parts of the body; while a large amount, by oxidation to carbonic acid and water, sets free energy which may either take the form of work or heat. The nature of fatty degenerations and infiltrations of organs will be referred to under the special pathological headings.

Absorption of Putrefaction Products.—The products of putrefaction formed in the lower parts of the intestinal canal may also be absorbed. This is especially noticeable in cases of intestinal obstruction, when phenol, indol, skatol, etc., are absorbed, oxidised in the tissues and excreted in combination with sulphuric acid.

Intestinal Movements. — It is exceedingly difficult to study the normal peristaltic movements of the small and large intestines, because on exposing them to the air there is a marked diminution in the peristalsis. They have been usually studied when the intestines have been surrounded by a warm saline solution. One can then perceive that peristalsis is more marked in the small than in the large intestine, and that it takes place normally in the direction towards the anus, the movements consisting of ring-like contractions passing along in the form of waves. The correct direction is maintained by the transverse folds of the intestinal mucosa acting as valves, the well-marked fold at the place of entrance of the small intestine into the cæcum preventing regurgitation. Antiperistaltic movements, such as occur in fæcal vomiting, are not to be seen under normal conditions. The fact that this peristalsis proceeds in one direction only, and that slowly, proves that it must be under the influence of the nervous system, and yet it persists after section of the mesenteric nerves, being also visible in separated intestinal loops. It is probable, therefore, that there are local centres present in the intestinal wall. The nerves passing to the intestine act as regulators of the movement, probably affecting these local centres. Thus stimulation of the vagus increases the rapidity or amplitude of the contractions, while splanchnic stimulation produces slowing of the peristalsis. If indifferent fluids be circulated through the vessels of a detached loop of intestine, it remains at rest; but, whenever the circulation stops, the loop shows periodic contractions. The explanation of this is that in the intestinal wall substances are formed which act as excitants to contraction, and hence their removal produces rest. The contraction of intestinal blood-vessels produced by splanchnic stimulation probably causes diminished peristalsis from the diminution in the amount of circulating blood which contains the stimulating substances. There is probably a reflex nervous mechanism between stomach and duodenum, because shortly before the pylorus opens, the duodenum, which was previously at rest, begins to contract. The following conditions also affect intestinal peristalsis.

1. *Alterations in temperature.*—On exposing the intestines to a temperature below 7° C., peristaltic movements stop, while on raising the temperature they increase. At 39° C. peristalsis in the rabbit's intestine ceases, beginning again when the temperature is raised to 42° C.

2. *Effects of food and drink.*—About fifteen minutes after food or after a drink of cold water peristalsis begins, while during the night the movements are markedly diminished.

3. *Effect of drugs.*—Opium and atropin paralyse the action of vagus, and so hinder peristalsis; while nicotine, both after injection into a blood-vessel and into the lumen of the intestine, produces increased contractions. Potassium and sodium salts also produce contractions, the former local, the latter more general in character.

4 *Action of intestinal contents.*—The bile and the intestinal gases increase peristalsis.

FÆCES.—The fæces contain the undigested residues of food-stuffs, decomposition products, such as indol, formed in the intestine, material secreted or shed by the passages through which the food has passed and micro-organisms of different kinds. As a rule their reaction is acid, though it may be neutral or alkaline, and their consistence also varies greatly according to the nature of the food and the time spent in the intestinal canal. The food may act upon the character of the fæces either directly from its own composition or from its effect upon the gland secretions which are poured into the canal or from its action on intestinal peristalsis. Thus a vegetable diet, from the high percentage of indigestible material and of water, and from the stimulant action of the insoluble cellulose on intestinal peristalsis, produces excreta which are characterised by their fluid consistence, richness in total solids, and, as a rule, poverty in colouring material. Owing to the short period that the material is allowed to lie in the canal, reduction processes do not take place to the same extent as after a flesh diet. If the vegetable diet be rich in iron, the fæces are much darker in colour. The dark colour after flesh diet is due to the presence of hæmatin or bodies derived from it (ferrous sulphide, etc.), and also to reduced bile pigments or bodies derived from them. Under a mixed diet about 130 grammes are excreted in twenty-four hours. The most important gases present are nitrogen, hydrogen, carbonic acid, sulphuretted hydrogen, and methane. Fæces can be formed in a separated loop of intestine, and also in the intestinal canal of starving animals even when bile has not been allowed to enter the intestine. The excreta are formed in such cases from the shedding of epithelium and the subsequent decomposition of such organic material by bacteria. Different bacteria may be found in the fæces, *e.g.* putrefaction and fermentation organisms and bacillus coli communis.

DEFÆCATION.—Although the act is a reflex one, carried out through a centre in the spinal cord, it is largely influenced by the will. In man this centre is situated in the lumbar region of the cord. The pressure of the accumulated fæces in the rectum causes, by reflex action through the centre, relaxation of the sphincter, while increased intestinal peristalsis is also set up. In voluntary defæcation, the act is started by a full inspiration, closure of the glottis and fixation of the diaphragm, followed by contraction of the abdominal muscles and levator ani, the latter in this way exerting pressure on the rectum while the tension of the pelvic fascia offers the required resistance. By an effort of the will the act may, up to a certain point, be inhibited especially by increasing the contraction of the sphincter. Purgatives may act in different ways. The activity of salines depends upon their endosmotic equivalent, producing a retention of water in the intestine. Thus if salines be injected into the blood-vessels, constipation results. Some again act by increasing the amount of the intestinal secretions, while others act by increasing the rapidity or strength of the intestinal contractions (see "Constipation").

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Supplemental Notes.